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A STANDARD ELECTROENCEPHALOGRAPHIC TECHNIC FOR THE LOCALIZATION OF GROSS INTRACRANIAL LESIONS*

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SINCE Walter[1] first reported his technic for the localization of intracranial lesions by electroencephalography, ample evidence[2–8] has accumulated to prove beyond question its value as a supplement to previously available methods. The present report describes and gives the results obtained with a standardized and simplified technic, which, it is believed, makes electroencephalographic localization a more practicable clinical procedure. Except for a master selector switch, which will be described later, the required apparatus is the same as that used by Williams and Gibbs.[4, 5]

APPARATUS

The apparatus consists of three channels of undistorted amplification of all frequencies from 1 to 1000, with a voltage intensification of approximately ten million times, a noise level of less than 5 microvolts, and three ink-writing oscillographs that record without distortion up to 80 cycles per second. The amplifiers should be so designed that they are independent of each other, that is, the signals on one channel should not affect the signals on either of the other channels.

One of us (F. A. G.), in previous reports with Williams,[4, 5] subscribed to the use of cotton pledget electrodes wet with salt solution and held on the head with elastic bands. Such electrodes have the great advantage of being easily placed in any desired combination of positions, but they are so liable, by shifting and drying, to produce artifacts resembling the disorder produced by a tumor that it seemed hazardous to use them in ordinary clinical practice. If the type of electrode introduced by E. L. Gibbs is used, electrode artifacts can be greatly reduced or eliminated. Such electrodes are made by flattening a solder pellet on the end of a fine (34-gauge) enameled wire. This can be done by thrusting the bared end of the wire into a heated drop of solder and pressing on the drop while still hot. The flattened pellet should form a disk about 1 or 2 mm. thick and approximately 5 mm. in diameter. Contact with the scalp is made by covering one side of the electrode with a saline paste (Sanborn electrode paste). A detailed description of the precautions to be taken in applying such electrodes is given elsewhere.[9]

In localizing intracranial lesions, it is desirable to cover all accessible surfaces of the brain with electrodes. As a practical compromise, sixteen leads are sealed to the scalp and forehead, all more or less equidistant from each other (Fig. 1). They are arranged in four longitudinal columns in such a way that the first column, referred to as A, starts with Electrode 1 slightly anterior to the right temple; Electrode 2 is placed over the right inferior precentral area, Electrode 3 over the right inferior postcentral area and Electrode 4 halfway between the third and the right occipital pole. The next column, referred to as B, has Electrode 1 half way between the upper and lower limits of the forehead and directly over the pupil; Electrode 2 is in the right precentral region, about two thirds of the way from the mid-line to Electrode 2 in Column A, Electrode 3 in the postcentral area about two thirds of the way from the mid-line to Electrode 3 in Column A, and Electrode 4 slightly to the right of the occipital pole. Column C is the left-hemisphere equivalent of Column B, and Column D is the left-hemisphere equivalent of Column A.

Each electrode is connected by its 34-gauge enameled wire lead to a separate binding post on a

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junction box. The junction box is equipped with a master selector switch designed and constructed by A. M. Grass. By means of this switch, it is possible to connect the amplifiers to any four electrodes, longitudinal or transverse, as shown in Figure 2.

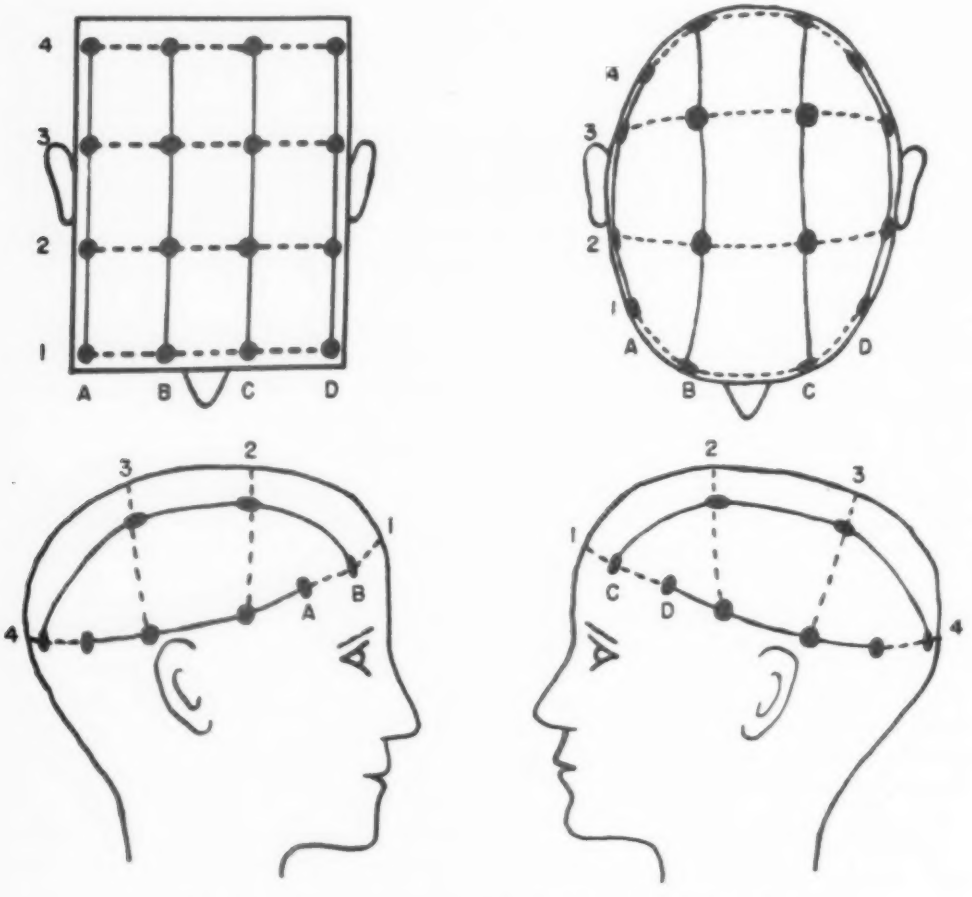

FIGURE 1. *Diagrams Showing the Position of the Electrodes.*

The diagram at the upper left shows the arrangement of electrodes on the schematized surface of the head, considered as a rectangle, with electrodes arranged in four equidistant columns, A, B, C and D, of four electrodes each and in four equidistant rows, 1, 2, 3 and 4, of four electrodes each. The other diagrams show the concessions that are made to the curved surface of the head in actual practice. The arrangement of the columns and of the rows is kept as nearly like the rectangular scheme as possible. The position of each electrode is described in the text.

CRITERION FOR LOCALIZATION

The criterion used for localization of gross organic lesions is that described by Walter,[1] namely, apparent reversal in phase of waves with a frequency of approximately ½ to 3 per second. Waves off this frequency Walter calls "delta waves." A region showing the maximal number and voltage of such waves (indicated by out-of-phase relations) he calls a "delta focus." Good illustrations of such foci are shown by Walter and by Williams and Gibbs.[5] Slow waves such as are seen around an organic lesion are more or less sinusoidal and very often have some normal activity superimposed on them. In general, the severer the lesion, the less the superimposed activity, the slower the waves and the higher their voltage. Extraneous voltage fluctuations, which might be confused with the slow waves from around a gross lesion, are discussed elsewhere.[9]

It is important to recognize at the outset that the apparatus cannot distinguish accurately between different types of lesion and that it cannot localize deep cerebral and subtentorial lesions. Smith and his co-workers[10] have said that subtentorial lesions can be localized with the electroencephalogram, but this is qualified to mean that

cases with cerebellar tumors show slow-wave foci in the occipital cortex. In our experience, such foci are usually lacking. Certain wave formations are commonly encountered in epilepsy. These have been discussed and illustrated elsewhere.[9] As previously stated, they are not generally satisfactory for localizing gross disease. However, any focus of epileptic discharge should be noted, for its presence and position — when taken with the rest of the clinical and laboratory evidence — may be a valuable diagnostic clue.

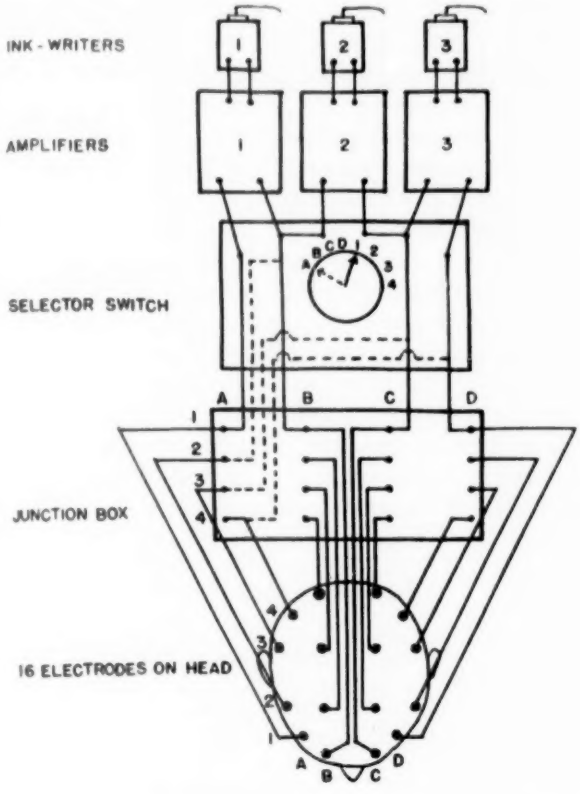

FIGURE 2. *Schematic Diagram of the Manner in Which the Electrodes are Connected.*

By turning the dial on the master selector switch to any one of the indicated positions, A, B, C, D, 1, 2, 3 *and* 4, *it is possible to connect the corresponding column or row to the amplifiers. When turned to a column (as shown by the dotted lines),* Electrodes 1 *and* 2 *go to Channel* 1, Electrodes 2 *and* 3 *to Channel* 2, *and* Electrodes 3 *and* 4 *to Channel* 3; *when a row is connected (as shown by the solid lines),* Electrodes A *and* B *go to Channel* 1, Electrodes B *and* C *to Channel* 2, *and* Electrodes C *and* D *to Channel* 3.

If a focus has been localized to the region under one electrode, the electrode should *always* be replaced with another close to it to make sure that a bad contact is not producing the slow waves.

If a focus cannot be detected with the primary placement of electrodes, and if the clinical signs point to a specific area, all sixteen electrodes should be placed so that they form a square covering and extending beyond the presumable site of the lesion.

When the electroencephalographic localization is the sole or chief reason for contemplating surgical exploration of a particular area, it should be repeated on a subsequent day, with the sixteen electrodes concentrated around the region in which the focus was previously localized, to make sure that the focus is still there and to delineate it more sharply.

By concentrating the electrodes in a particular area, it is possible to get the maximum accuracy obtainable with electrodes placed on the outer surface of the skull. Grinker and Serota[11] have used electrode placements on the inferior surface of the cranium, and Jasper[12] has employed these electrode placements for localizing tumors. In certain cases, these so-called "nasal electrodes" may give important evidence. At any rate, the inferior surface of the brain is of interest in many clinical problems and should not be disregarded. However, temporal-lobe tumors and tumors arising from the sphenoidal ridge can be localized with electrodes on the outer surface of the head. For clinical studies, it seems best to attempt first a localization with the sixteen electrodes covering the outer surface of the head and later, if additional evidence is required, to use special electrode placements, which are adjusted to the exigencies of the particular situation.

TABLE 1. *Results of Electroencephalographic Examination of 144 Patients Suspected of Having Operable Intracranial Lesions.*

RESULTS		No. OF PATIENTS
Examination revealed no focus		91
Discharged — insufficient evidence of operable lesion	83	
Operated on	8	
No gross lesion found	5	
Deep cerebral or subtentorial lesion found	3	
Examination revealed no focus		53
Discharged — insufficient evidence of operable lesion	15	
Refused operation	3	
Operated on	35	
No gross lesion found	5	
Gross lesion found	30	
Focus not in same region as lesion	3	
Focus in same region as lesion	27	
Total		144

RESULTS

The results of the electroencephalographic examination of 144 patients suspected of having gross intracranial lesions are shown in Table 1. In 33 patients with lesions found at operation, the neurologic examination led to a correct localization in 64 per cent, x-ray study without air in-

jection in 21 per cent, x-ray study with air injection in 39 per cent and electroencephalography in 82 per cent.

The poor score for air injection followed by x-ray examination is not owing to any inaccuracy of the procedure but merely to the fact that because of discomfort to the patient, danger or expense, it was not used in the majority of cases. The true accuracy of the method appears when one considers the 14 cases in which it was used; it led to a correct localization in all except one case, that is, it was 93 per cent accurate. In the 10 cases in which no lesion was found at operation, a localized lesion was indicated by the neurologic examination in 7, by x-ray study without air injection in 2, by x-ray with air injection in 5 and by the electroencephalogram in 5.

Discussion

It is unlikely that any electroencephalographic method of localizing space-consuming lesions will give results as good as x-ray study with air injection, for the latter gives almost direct evidence of the lesion. On the other hand, electroencephalography is painless, without danger and relatively cheap, so that there is no reason why it cannot be used on all cases. It should be regarded as a special type of neurologic examination, which has the following advantage over the ordinary neurologic examination: it can test the functional activity of various cortical areas that are clinically silent. In the cases in this series that had gross supratentorial lesions, electroencephalography was more useful than urologic examination for localization. The comparative values and different methods of localization are discussed here to show how useful electroencephalography is in terms of other technics now commonly employed, not with the idea that any of the latter should be abandoned. Accurate localization of differently situated lesions of various types require the use of all available methods. Each method contributes to the total accuracy.

Summary and Conclusions

A standardized and simplified form of Walter's technic for the localization of gross intracranial lesions is described, and the results obtained with it on a series of 144 patients suspected of having an expanding intracranial lesion are given. Of the 98 patients discharged because of insufficient evidence of an operable lesion, 85 per cent showed no electroencephalographic evidence of localized gross disease. Among the 44 patients operated on, electroencephalography gave correct localizations or correct negative findings in 73 per cent. In the 30 cases in which the lesion involved the cerebral cortex, localizations were accurate in 90 per cent.

The technic described is useful for providing evidence of the presence or absence of gross cortical lesions and for localizing such lesions.

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TEMPORAL ARTERITIS*

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UNTIL recently, the diagnosis of arteritis has been made in most cases by the pathologist. For this reason, the impression was gained clinically that all cases of arteritis and periarteritis were necessarily fatal. More careful observation of the superficial vessels has shown that arteritis and periarteritis are not only commoner and more widely distributed than was formerly believed, but that, in addition, the prognosis in certain types is distinctly favorable. During the last few years, arteritis of the superficial temporal vessels has emerged as a distinct clinical entity in which the prognosis is entirely favorable, even though the pathologic changes are indistinguishable from other types of arteritis. It has therefore seemed appropriate to report the cases of 3 patients who recovered from acute temporal arteritis, the diagnosis having been proved by biopsy.

CASE REPORTS

CASE 1. M. B., a 58-year-old married woman, entered the hospital on July 2, 1940. She had been well until 3 weeks before admission, when she developed a severe headache along the right side of the forehead, the pain going down the right side of the neck. The pain soon spread to involve both sides of the head and neck, extending up to the top of the head and across the forehead. There was also a severe pain behind the eyes, and a marked paresthesia of the scalp, so that it was impossible for the patient to comb her hair. The past and family histories were noncontributory.

Physical examination on admission to the hospital revealed tenderness over both temporomandibular joints. It was impossible for the patient to open her mouth completely. There was some redness of the throat, the anterior pillars being more inflamed on the right than on the left. Small deep cervical lymph nodes were palpated on both sides, but no other lymphadenopathy was noted. There was exquisite tenderness over the temporal arteries on both sides. The heart, lungs, abdomen and reflexes were normal. The temperature was 101°F., the pulse 80, the respirations 20, and the blood pressure 118/62.

Laboratory examinations revealed negative blood Wassermann and Hinton reactions. The urine was normal. During the patient's 35 days in the hospital, the temperature varied from 98 to 103°F., with a tendency toward an afternoon rise. The white-cell count varied between 10,000 and 20,000, the majority of counts being approximately 14,000. Differential counts revealed 76 to 80 per cent neutrophils; the highest eosinophil count was 4 per cent, noted on only one occasion. The sedimentation rates were 62 and 70 mm. in 1 hour by the Wintrobe method, the normal rate being 20 mm. The uric acid and nonprotein nitrogen levels of the blood were within normal limits. Agglutination tests for typhoid fever, paratyphoid (A and B) fever, brucellosis and tularemia and a sheep-cell agglutination test were negative, as was a gonococcus complement-fixation test.

Because of the possibility of meningitis, at the time of admission, a lumbar puncture was performed; the spinal fluid showed normal dynamics and pressure readings, a protein of 20 mg. per 100 cc., and 1 cell per cubic millimeter.

During the patient's stay in the hospital, she developed marked tenderness and induration over both temporal arteries, but no nodules. The severity of the tenderness changed from day to day and varied on both sides, as did the swelling over the temporomandibular joints. The frontal headache persisted, but could be alleviated by pressure over the temporal artery on either side. Codeine, morphine, Nembutal and phenacetin were used for temporary alleviation of the pain, but were not effective. On the 19th hospital day, sulfapyridine was begun, was continued for 4 days, and was discontinued because of marked nausea, vomiting and generalized discomfort. A free-sulfapyridine level of 7.4 mg. per 100 cc. was obtained, but there was no change in the patient's general condition or symptomatology.

FIGURE 1. *Photograph of Biopsy Specimen.*

This section shows the thickened wall and the narrowed lumen of the artery from Case 1 (eosin-methylene-blue stain, × 55).

On the 28th hospital day, a biopsy of the right temporal artery was performed under local anesthesia. On gross examination, the section showed some inflammation around the site of the temporal artery, and the artery

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appeared indurated. The pathological report was as follows:

> The lumen of the vessel is diminished in caliber as a result of proliferation of the intima [Fig. 1]. The lumen is elongated, slender and lined by a single layer of endothelium, beneath which there is a large amount of fibrous tissue without inflammatory cellular infiltration. The internal elastic membrane is somewhat fragmented, but is not reduplicated. Within the muscular coat, chiefly in the internal longitudinal layer and in the outer circular layer of the media, but extending also into the adventitia, there are many small foci of cells resembling those of a granulomatous lesion. This area displays a central necrosis, with fibrinoid degeneration. There are numerous multinucleated giant cells and large monocytes at the periphery of the section [Fig. 2]. In addition to these monocytes, the peripheral zone shows many lymphocytes mingled with some eosinophilic leukocytes and a few neutrophilic polymorphonuclear leukocytes. Careful search with various stains failed to reveal any micro-organisms. There is some increase in the fibrous tissue of the adventitia, and the smaller vessels in the surrounding fat are congested. Otherwise the fat and the striated muscle about the vessel are not remarkable.
>
> The lesions in this vessel correspond to those described in cases of so-called "periarteritis nodosa" of the temporal artery. In our opinion, however, they bear more resemblance to the lesions in thromboangiitis obliterans (Buerger's disease) than they do to periarteritis nodosa.

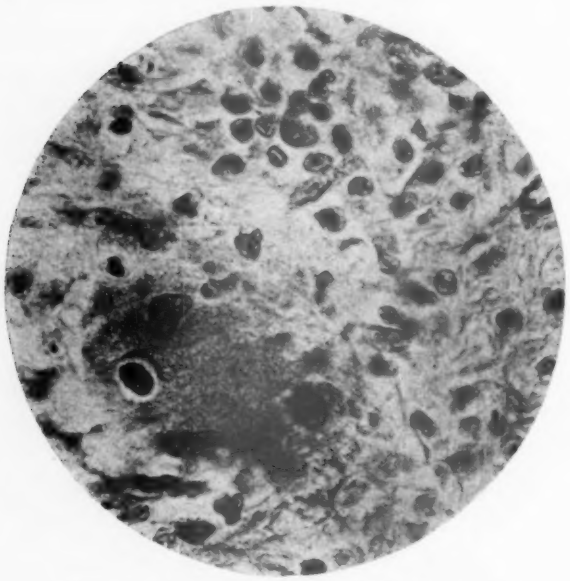

FIGURE 2. *Photograph of Biopsy Specimen.*

This section shows a giant cell and the inflammatory infiltration in the wall of the artery from Case 1 (eosin-methylene-blue stain, × 640).

Within a few days following the biopsy, the headache, paresthesia and tenderness were relieved, but the elevated temperature, increased sedimentation rate and leukocytosis persisted. The patient was discharged shortly after the biopsy was performed, and at present is symptom free. The blood counts and the temperature are normal. The sedimentation rate, although still elevated, shows a tendency to be lower.

CASE 2. H. M., a 61-year-old widow, entered the hospital on April 20, 1939. Her complaints at that time were pain in the shoulders, hips, knees and back of 3 weeks' duration, and pain, swelling and tenderness around the temporomandibular joints of 2 days' duration. She also complained of severe generalized throbbing headache of about 3 weeks' duration. The past history and the family history were noncontributory.

When the patient entered the hospital, the temperature was 100°F., the pulse 82, and the respirations 20. The blood pressure was 130/80. Physical examination revealed that the mandibular joints were tender, and that motion was limited by pain and swelling under both jaws. There was swelling of the lymph nodes in both submaxillary areas, but no other lymph nodes were enlarged. There were slightly red, indurated and tender areas over both temporal arteries. Pressure over the temporal arteries relieved the generalized frontal headache.

The blood Wassermann and Hinton reactions were negative. The urine was clear, and showed no albumin, sugar or casts. The white-cell count varied from 12,000 to 14,000, with 80 per cent neutrophils. Three per cent eosinophils were recorded on one occasion. Blood culture was negative. A skin test for tuberculosis with purified protein derivative of the first strength was negative. The basal metabolic rate was +12 per cent. The icteric index and an electrocardiogram were normal.

During the course of the illness, the patient remained comfortable on large doses of acetylsalicylic acid. The temperature varied from 99 to 101°F. The patient received a course of sulfanilamide for 8 days, during which she developed a marked gastrointestinal upset and an anemia. The red-cell count fell to 2,700,000. However, no essential change in the headaches or febrile course was noted. X-ray examination of the spine showed a moderate degree of arthritis.

On the 27th hospital day, a biopsy of the right temporal artery was taken. Microscopic examination of the section was reported as follows:

> The artery shows marked chronic inflammation in all its coats. It is surrounded by chronic granulation tissue in which well-developed capillaries are seen, and in which proliferating fibroblasts are scattered from place to place. The entire area is densely infiltrated with cells that are predominantly lymphocytes, with a large number of monocytes, scattered polymorphonuclears, a rare eosinophil and occasional multinucleated cells that might be designated giant cells. A few lymphocytes extend into the muscular coat, separating the muscle bundles. The muscular coat is slightly thinned, and throughout its margin there are irregularly shaped masses of basophilic material, presumably fibrin. The intima is composed of proliferating connective-tissue cells, and only a minute lumen remains.

The headache disappeared to some extent after biopsy, but the patient was still febrile when she was discharged on the 32nd day.

A follow-up examination a year later revealed that the patient's elevated temperature and headache had almost completely disappeared, but she still complained of pain in the shoulders, hips, knees and back.

CASE 3. P. N., a 67-year-old store manager, was seen in the Out-Door Department on September 26, 1939. At that time, he complained of soreness and muscular aches

all over his body of 3 months' duration. The patient also complained of generalized headache, pains around the mandibular joint and down the sides of the neck of 3 months' duration.

The past history was significant in that the patient had had asthma, presumably on an infectious basis, for 5 or 6 years before this visit.

Physical examination revealed both temporal arteries to be enlarged and painful. The temperature was 98°F., the pulse 72, and the blood pressure 195/100. The rest of the examination was not remarkable. Without being referred into the hospital, the patient was anesthetized locally, and a partial excision of the left temporal artery was performed. The pathologic description of the section was as follows:

> There is marked reduction of the lumen, owing to intimal proliferation. Peripherally, the intima tends to be of a collagenous character, whereas centrally it is more cellular and fibroblastic. A few lymphocytes and eosinophilic leukocytes are present throughout the intima. The internal elastic membrane is prominent. At some levels it completely surrounds the intima, and at others it becomes imperceptible in an area of fibroblastic proliferation and inflammatory cell reaction. Throughout most of the circumference of the artery, the normal parallel architectural arrangement of connective tissue and smooth muscle fibers of the media is well preserved. In some areas, this arrangement is obliterated by an intense lymphocytic infiltration, accompanying which there is a marked multinucleated giant-cell reaction.

Postoperative recovery was uneventful.

When the patient was last seen, 1½ years later, the headache and pain around the mandibular joints had practically disappeared.

Discussion

Historically, acute temporal arteritis has been mentioned in the literature since 1931. At that time, Horton, Magath and Brown[1] published a report of 2 cases. Since then, 16 additional cases have been reported.[2–8]

There is considerable similarity between the cases reported in the literature and our 3 cases. All the 19 patients reported have been elderly people, ranging in age from fifty-five to eighty years. The majority of the patients have been women. The presenting symptom is usually a severe, boring ache referable to both sides of the head, although usually severer on one side than on the other. The pain also seems to be severer at night, so much so that it prevents the patients from sleeping. In our experience, the headache is so intense that it is unrelieved by morphine or barbiturates. The temporal regions and temporal arteries may be tender, and during the course of the illness, the vessels may become enlarged, tortuous and surrounded by areas of hyperemia. Tenderness of the scalp and painful mastication may also be present—particularly prominent features in Case 1. In addition to the headache, the patient may complain of severe generalized symptoms. These patients often present the appearance of being severely ill, entirely out of proportion to the amount of local disease present. It is possible that the inflammation of the temporal artery is but a local manifestation of a more widespread disease.

The pathologic findings of the cases reported above are similar to those previously described. The general lesion is one of chronic periarteritis and arteritis. There are areas of granulation tissue in the adventitia of the blood vessels that suggest granuloma. There is an infiltration of round cells in the adventitia around the vasa vasorum, and to a marked extent in the media of the vessels. There may be a complete necrosis of the media, which is sometimes replaced by a granulomatous type of lesion. In all the cases in the literature and in our own cases, giant cells were present, and this seems to be a most constant factor. The lumen may be reduced in size, and nodular areas may appear along the outer aspect of the vessels.

From a practical standpoint, the most effective treatment seems to be the removal of a segment of the temporal artery. This has been demonstrated in the 3 cases presented, and in 4 other cases in the literature.[3, 4, 7, 8] After section of the artery, the headache is relieved, even though the fever and generalized symptoms may continue for as long as several weeks. It has been suggested that section of the artery severs the sympathetic nerve fibers,[8] and in this way eliminates the pain, although it is difficult to explain the ultimate relief of generalized symptoms with this treatment. It is worth noting that, in Cases 1 and 2, pressure over the temporal artery reduced the severity of the headache. This may be a point in determining whether section of the artery is going to be beneficial.

Because of the extreme practical importance of an accurate diagnosis before biopsy is undertaken, other common causes for the severe symptoms should be differentiated. First, there are intracranial causes, such as brain tumor and meningitis, and secondly, extracranial causes, such as phlebitis of the scalp veins, osteomyelitis of the malar and temporal bones, sinusitis, subcranial infection and herpes zoster. Biopsy of the temporal artery is justified in a patient with headache, fever, malaise, exquisite tenderness over the temporal artery and no other evidence of disease.

The cause of this localized arteritis is not known. Thromboses of the vasa vasorum might be an explanation,[8] but the reason for thromboses in this area is not apparent. Bacteriologic studies have been negative in all biopsy specimens. The fact that the pathologic specimens demonstrate

only acute and chronic inflammation leads one to suspect that this is a localized form of periarteritis, fortunately in an accessible place and in a vessel not essential to life. It might be well that all knowledge referable to arteritis of the temporal vessels be retained as an entity until further studies elucidate the exact relation of this clinical syndrome to other types of arteritis.

Summary and Conclusions

Three cases of inflammation of the temporal artery, with pathologic reports, are presented. Arteritis of the temporal vessels appears to be a clinical entity with a favorable prognosis, although pathologically it is not distinguishable from other types of arteritis. Biopsy was distinctly beneficial in alleviating the patients' symptoms.

I am indebted to Dr. Orville Bailey of the Pathological Department for preparing the photomicrographs.

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ELECTROCARDIOGRAPHIC FINDINGS ASSOCIATED WITH A GUNSHOT WOUND OF THE HEART*

Report of a Case

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OPERATIVE wounds of the heart offer the unique opportunity to study the effects on the electrocardiogram of trauma of known character, severity and location. Great care, however, must be exercised in ascribing given changes in the electrocardiogram to the injured heart. The several factors, each of which may contribute to the electrocardiographic pattern, include: the previous normal pattern of the patient's electrocardiogram, pericarditis, pericardial effusion, tamponade, pneumothorax with mediastinal shift, and myocardial damage (degeneration, infarction or fibrosis).

Characteristic electrocardiograms have been described[1-3] for cases of pericarditis, myocardial infarction and the combined effect after experimental ligation of a coronary artery. An analysis of cases of wounds of the heart[4] showed differences in the effect on the RST segment that were related to the presence or absence of involvement of the left coronary artery. Similarly, T-wave inversions have been found to differentiate cardiac injuries with associated coronary-artery involvement and those with pericarditis alone as the principal factor affecting the electrocardiogram. If the preponderant effect on the electrocardiogram is owing to pericarditis, inversion of the T waves in the three standard leads and the reciprocal loss of elevation and depression in the ST segments in Leads 1, and 3 may be expected.

The present report of a gunshot wound of the heart, involving a branch of the artery to the anterior and lateral portions of the left ventricle that arose from the left circumflex artery, presents the electrocardiographic changes recorded preoperatively, during the course of operation and postoperatively. It must be emphasized that the records represent not only the effects of the cardiac injury but also the attendant complications, which included hemopericardium, pericarditis, pneumothorax and pleural effusion, as well as trauma incident to manipulation during the surgical procedure.

Case Report

A 19-year-old housewife was brought to the Knickerbocker Hospital via ambulance on March 30, 1940, after having accidentally fired a 25-caliber revolver bullet into her precordial area. On admission to the hospital, the patient was bleeding from the nose and mouth and was in marked shock, the pulse rate being 120, the respirations 24 and grunty in character, and the blood pressure 90/60. The point of entrance of the bullet was in the 4th left intercostal space, 1.5 cm. from the sternal margin. The point of exit was situated posteriorly in the same horizontal and sagittal planes as the entering wound. There was no active bleeding from the external bullet wounds. The heart sounds were of fair quality. No thrills or murmurs were present. The rhythm was rapid and regular but frequently interrupted by premature contractions. Exam-

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†Associate professor of surgery, New York Medical College; surgeon, Knickerbocker Hospital.

‡Associate physician and physician-in-charge of the Cardiac Clinic, Knickerbocker Hospital.

ination of the lungs revealed dullness to flatness over the entire left chest.

The immediate treatment included external heat, intravenous physiologic saline solution and morphine. Tetanus antitoxin and gas-gangrene antiserum were also administered.

At operation, the edges of the wound of entrance were blackened with gunpowder. The subcutaneous tissue was markedly hemorrhagic. Under ether-oxygen anesthesia a 12-cm. curvilinear incision, with its convexity medially, was made from the 3rd to the 5th ribs at the sternocostal margin, and the skin, subcutaneous tissue and pectoralis muscle were reflected from the sternal margin. The sternocostal joints of the 3rd, 4th and 5th ribs were next incised, and the ribs reflected and retracted laterally. Bleeding from the intercostal and internal mammary vessels was controlled. The pleural cavity was then entered, and the collapsed lung packed off laterally. The heart lay exposed following incision of the pericardium.

The pleural cavity contained between 300 and 500 cc. of bright-red blood, and the pericardial sac was filled and distended with fresh and partially clotted blood. A roughly oval rent (2 by 1 cm.) was encountered in the pericardial sac. A jagged laceration was noted in the myocardium on the obtuse margin of the heart, approximately 5.0 cm. lateral to the left anterior descending branch of the left coronary artery and about 2.5 cm. inferior to the auriculoventricular junction. The myocardial laceration had penetrated the left ventricular chamber so that an opening 0.5 cm. in diameter was noted when the clot, which acted as a tampon, was released, after the pericardial sac was opened. In addition to the myocardial injury, a laceration of the obtuse branch of the left coronary artery was found.

The left index fingertip controlled the ventricular bleeding while three stay sutures of No. 00 black vaselined silk were inserted at the lower angle of the myocardial laceration to close the opening in the heart wall. A ligature was then placed about the lacerated coronary vessel, and the site reinforced by a small piece of fat removed from the subcutaneous tissue. After ascertaining that no further bleeding was occurring, the pericardial sac was closed with a continuous suture of No. 00 black silk. The pleura, intercostal spaces and pectoralis muscle were repaired with interrupted No. 1 and No. 5 black silk sutures, and No. 00 black silk sutures were used in the subcutaneous tissue and skin. No drains were used. The sites of the bullet's entrance and exit were excised and repaired with No. 00 black silk sutures. The operative procedure was begun about 1½ hours after the injury and required 40 minutes.

The patient's blood pressure was 80/30 at the start of the operation, whereas at its conclusion it was 90/40. The pulse throughout was of good quality and regular, and occasionally showed pauses compensatory to premature ventricular contractions. The pulse rate varied between 100 and 120. Respirations were regular and varied between 30 and 36. A 500-cc. transfusion of citrated blood was administered during the operation, and subsequently three additional transfusions provided a total of 1400 cc. of blood. Postoperatively, the patient was promptly placed in an oxygen tent. Consciousness was regained after 6 hours, when the blood pressure was 112/76, the pulse rate 120 and of good quality, and the respirations 36 and shallow. During the subsequent 48 hours, the patient received 10 gm. of sulfamethylthiazole as a preventive measure to combat infection. Fluids were well tolerated, and the urinary output was good. On the 3rd day, the sulfamethylthiazole was discontinued because the right upper extremity showed a flaccid paralysis, with wrist drop, and accordingly large doses of vitamin B_1 were administered parenterally. After 3 days, use of the arm was restored, but the wrist drop persisted for 4 additional days. The temperature rose to 106.5°F. on the 1st day and gradually fell to normal on the 10th day. On the 4th day, because of labored respiration, left-chest pain and physical signs of fluid, a thoracentesis was performed,

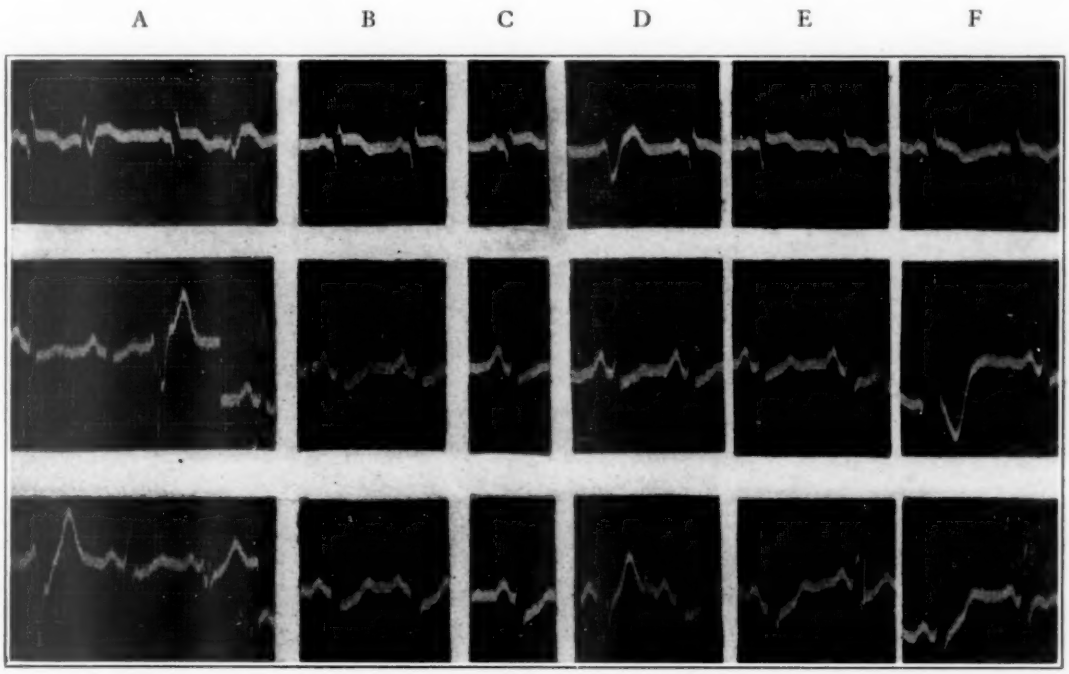

FIGURE 1.

and 700 cc. of dark-red blood aspirated. Three weeks later, a second thoracentesis yielded 600 cc. of dark-amber sterile fluid. The operative wounds healed by primary union. During the 5th week of the patient's illness, the temperature hovered about 101°F. but dropped to normal within 48 hours after a third thoracentesis, which yielded 900 cc. of clear, yellow sterile fluid. The subsequent course was uneventful, the patient being allowed out of bed on the 45th day and discharged on the 50th day.

X-ray studies of the chest taken on admission and subsequently on the 1st, 4th, 12th, 19th and 21st days postoperatively revealed progressive changes consistent with the accumulation and resorption and aspiration of the pleural fluids.

Electrocardiograms were taken preoperatively (Fig. 1*A*), during the operation (Figs. 1 and 2, *B-M*) and postoperatively (Fig. 3). The findings are discussed below.

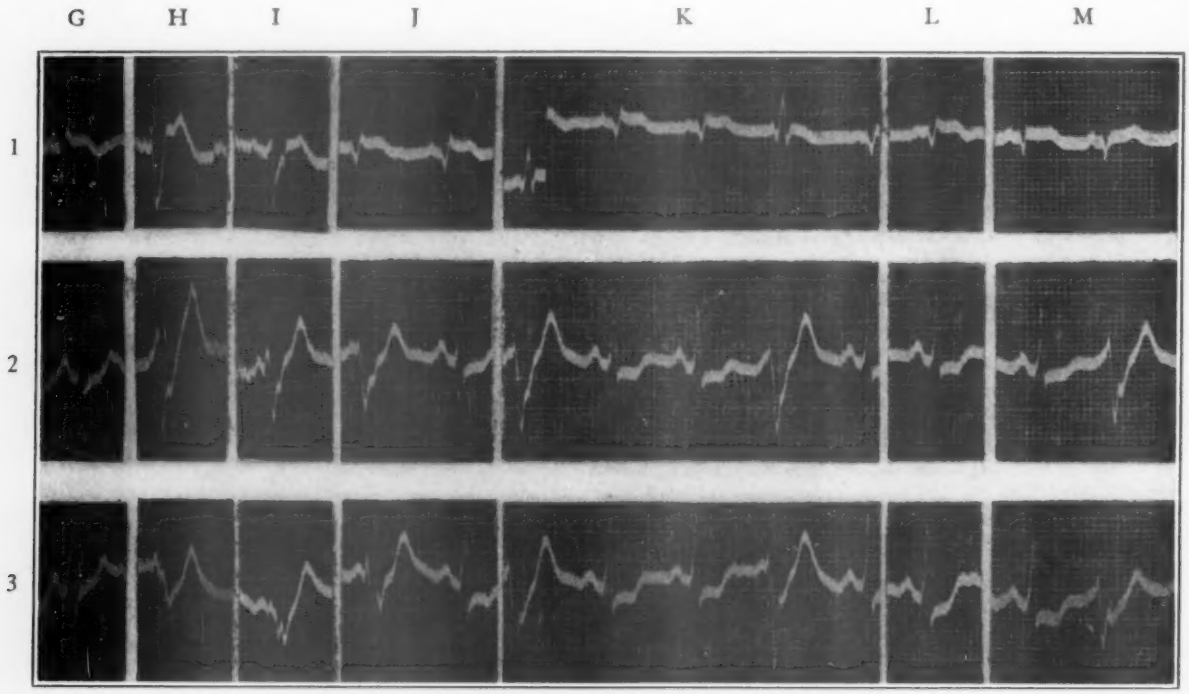

FIGURE 2.

ELECTROCARDIOGRAPHIC FINDINGS

Rhythm. The basic rhythm preoperatively and during the operation, as well as during the subsequent twenty-four hours, was a sinus tachycardia, with premature ventricular contractions and a ventricular rate of 120. After twenty-four hours, normal sinus rhythm ensued, with a rate varying between 80 and 100. Ventricular premature contractions were noted preoperatively and during the course of the operation. In Lead 1 of Tracing A, taken preoperatively, coupled rhythm is seen, each normal complex being followed by a ventricular premature contraction each of which shows a slightly different configuration suggesting a varying origin. In Lead 1 of Tracing D, the ventricular premature contraction has its main deflection downward, indicating a left ventricular origin, as shown originally by Barker et al.[5] and corroborated by Marvin.[6] This strongly points to an origin corresponding to the site of injury. The ventricular premature contractions in Lead 3 of Tracing A are of notably different patterns and probably represent two separate foci of origin. This type of wave is again noted in Lead 2 of Tracing F. A sinus rhythm with a ventricular premature contraction entering at each fourth beat is noted in Leads 2 and 3 in Tracing K, and a ventricular premature contraction at every third beat is seen in all leads of Tracing M.

Q wave. Tracing A reveals a deep Q wave in Lead 1. Beginning with a depth of 3 mm., it reaches a depth of 6 mm. in Tracing F, at the time of entering and aspirating the pericardial sac. The Q wave then recedes to a depth of 2 mm. when the chest cavity was finally closed (Tracings L and M). Subsequently, it remained between 1 and 2 mm., but was no longer 25 per cent or more of the main deflection. No other leads at any time showed an initial downward deflection.

Electrical axis. At the time of operation, when the deep Q wave was present (Tracing F), a right ventricular preponderance is noted. Subsequently, the deviation of the electrical axis became normal.

P wave. In Leads 1 and 3, in the tracings taken before and during the operation, the P waves are

2.0 to 3.0 mm. in height, becoming 1.0 to 1.5 mm. in height during recovery.

PR interval. The PR interval was 0.12 second throughout the period of observation.

QRS interval. The QRS interval in the normal complexes was 0.06 second throughout the entire period of observation.

ST segment. ST_1 is elevated in the tracings taken at the beginning of the observation, and

Lead 4. Preoperatively (no tracing shown), P_4 was inverted, S_4 prominent, and ST_4 depressed. Subsequently the P waves and ST segments became practically isoelectric, and T_4 small and inverted.

Summary

A bullet wound of the heart involving a branch of the left circumflex artery supplying the anterior and lateral portions of the left ventricle was noted

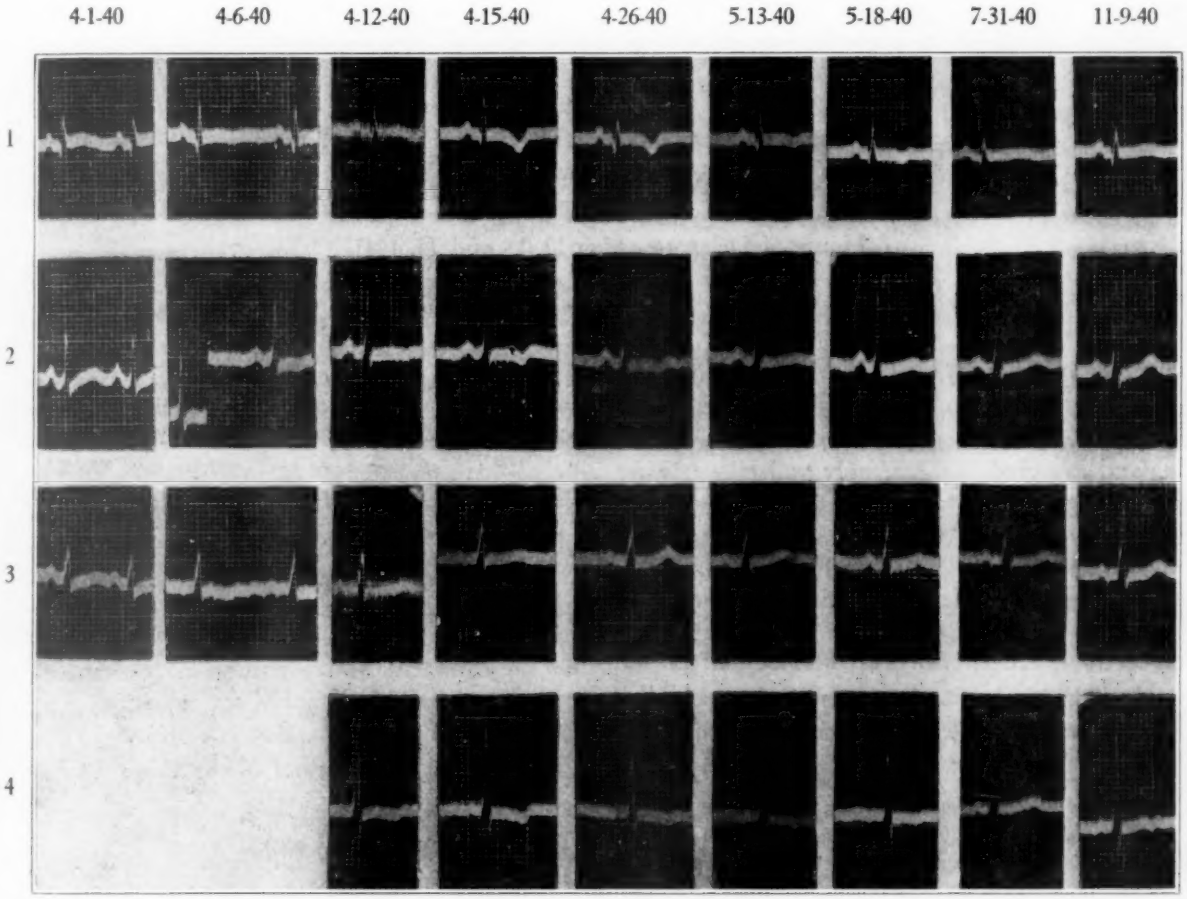

Figure 3.

remains so in those taken during and one day after operation. Five days later, it became isoelectric and remained so thereafter. ST_2 was depressed before and during operation (1.0 to 2.0 mm.). Postoperatively, it gradually reached a level about 0.5 mm. below the isoelectric line. ST_3 changes were parallel with those of ST_2.

T wave. T_1 was inverted and became most apparent as the ST segment gradually became isoelectric. T_2 was upright, except for a period of about one month beginning twelve days postoperatively, when it was inverted. T_3 remained essentially upright throughout, although the depressed ST_3 segment at times obscured the interpretation.

to be associated with the following electrocardiographic findings. Ventricular premature contractions arising in several foci were superimposed on normal sinus rhythm and sinus tachycardia. The premature contractions occurred both sporadically and at regular intervals, varying from coupling to tripling and quadrupling. The configuration of the premature contractions noted most frequently corresponded to that expected from the site of the injury. Q_1 became sufficiently deep at one period to cause deviation of the electrical axis to the right. No conduction defects were noted. ST changes corresponded to effects found in cases of coronary thrombosis with infarction.

T_1 became inverted — the most notable change.

It appears that in this case the preponderant effect on the electrocardiogram resulted from myocardial injury with necrosis, although definite pericardial disease was also known to be present.

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NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTIETH ANNIVERSARY

House of Delegates, May 12, 13 and 14, 1941

(Concluded from the issue of July 24)

The work of the committee was approved by the Committee on Officers' Reports, and Dr. D. G. Smith urged that members attend the New England Postgraduate Assembly and take advantage of the Commonwealth Fund Fellowships; the report was adopted.

Dr. Charles H. Dolloff then read the report of the Committee on Mental and Social Hygiene.

Report of the Committee on Mental and Social Hygiene

The Committee on Mental and Social Hygiene has no special recommendations to make this year, and believes the Society should be given the opportunity to catch up on what has already been recommended in years past.

Demands are constantly being made for enlargement of the mental-hygiene program, with the establishment of more clinics in the larger centers, but the influence of the Society does not appear to be brought to bear on those who would be able to furnish the necessary money. The need of an extension of social service as part of the prophylactic program is obvious, but languishes for lack of support.

One of the outstanding contributions to the program of mental and social hygiene is the survey sponsored by Ex-Governor Huntley Spaulding.

Mental hygiene and social hygiene are primarily public matters, and must stand or fall according to the amount of public support they receive. It would be of great help if the force of this large body of physicians, through a legislative committee, could be put behind the legislation required for these public projects.

Right now our state institutions caring for the mentally afflicted are suffering from the dearth of good physicians and other employees, owing to their inability to pay adequate salaries and wages.

A medical and surgical building is being constructed at the State Hospital, and the State School is about to open bids for the construction of another dormitory.

Charles H. Dolloff
Benjamin W. Baker
John B. McKenna

Dr. D. G. Smith, of the Committee on Officers' Reports, recommended that the Committee on Public Relations be asked to watch for and to support bills in the legislature that provide for the improvement and extension of the mental-hygiene program and moved the adoption of the report; it was so voted.

The next report was that of the Committee on Tuberculosis.

Report of Committee on Tuberculosis

"A Good X-Ray Is Your Doctor's Best Aid in Discovering Early Tuberculosis" is the theme of the fourteenth annual nation-wide Early Diagnosis Campaign conducted in April each year by the tuberculosis associations throughout the nation.

For several years in this campaign the chest x-ray has been stressed as the correct diagnostic method for finding tuberculosis in its early, most curable stages. But this year, the x-ray looms as a great defense weapon, for it is the recognized means of ensuring that tuberculosis is kept out of the military forces of the country.

In World War I the main reliance was the usual diagnostic procedure — inspection, percussion and auscultation. Today it is known that this reliance resulted in rejection and discharge of but one eighth of the tuberculous subjects called into the service. Is it any wonder that the expenditures for hospitalization, compensation and rehabilitation for tuberculosis among the veterans is today entering the second billion of dollars by the federal government?

Today, through the program of chest x-raying of national guardsmen and selective-service men in New Hampshire, the federal government has been saved at least $150,000 in eventual charges for tuberculosis.

Beginning with the induction of the 197th Regiment, Anti-Aircraft, at Concord on September 16, 1940, and up to March 1 of this year, a total of 3167 national guardsmen and selective-service men have been chest x-rayed at the Concord camp grounds, the state armories and the new induction center in Manchester. Of these men, 15 were rejected because of evidence of tuberculosis of the lungs, and several other men were deferred because of diseases of the chest revealed by x-ray study.

It is difficult to total the costs of this service because of the fact that the staff members of official and nonofficial health agencies carried out the program in co-operation with the induction agencies; yet it is estimated that the cost did not exceed $2500. This particular service is cited as an index of the tremendous value of the x-ray in searching out the smallest evidences of the presence of tuberculosis of the lungs.

The tuberculin test is more and more demonstrating its value as an aid in the diagnosis of tuberculosis. With rapid reduction in the number of positive reactors to the test, its value as a screen in sifting out those harboring infection with tuberculosis bacilli becomes increasingly evident.

The value of tuberculin testing and chest x-raying of positive reactors among contacts of tuberculous patients is well recognized.

More frequent use of the tuberculin test and chest x-ray by physicians in their offices and in the hospitals can materially aid in early diagnosis.

New Hampshire continues to maintain an enviable position with reference to its tuberculosis death rate. The reduction in rate has been persistent and phenomenal, from 97.8 per 100,000 population in 1920 to 28.47 in 1939.

Many factors no doubt have and are contributing to the control and advance toward eradication of the disease in New Hampshire — better living and working conditions, the cure of many tuberculous patients through more prompt and better treatment, and the reduction in numbers of new cases developing through the destruction of tubercle bacilli at their source among the afflicted.

The sanatoriums at Glencliff and Pembroke report encouraging increases in numbers of patients receiving pneumothorax treatment. Technical improvement has given better results and fewer complications. Gratifying effects continue to be evident in patients receiving chest surgery. The generous and whole-hearted co-operation of our chest surgeons and the hospitals has made possible arrest of the disease in certain patients for whom there could be but a future of chronic invalidism and ultimate disaster. This service has been performed at a minimal cost only through the generous spirit of co-operation already mentioned.

A difficult problem continues to exist in the necessity of providing for sanatorium treatment for tuberculous patients in accordance with present-day standards of strict bedrest over long periods of time. The infirmary facilities at the Glencliff Sanatorium have been taxed to the utmost. Empty beds are present in the cottage wards but cannot be used except for convalescent patients, because of their inadequacy for hospital treatment and care.

The grateful appreciation of all concerned is extended to the practicing physicians of New Hampshire for their increasingly effective co-operation, particularly their willingness to urge and provide chest x-ray films through the hospitals at a nominal cost for clinic cases. The officers and directors of the New Hampshire Tuberculosis Association, the examining physicians at the chest diagnostic centers, the field nurses, the patients and the public join in this expression of appreciation. It is keenly realized that this generous sympathetic understanding and assistance on the part of our physicians is due to the keen desire of the members of the New Hampshire Medical Society to hasten the eradication of tuberculosis among the people, and to confidence in the services of the chest diagnostic clinics.

Your committee urges more use of the tuberculin test, either the Mantoux, using protein purified derivate, or the Vollmer patch test. We urge this for all contacts with tuberculosis, for all patients presenting suspicious symptoms or signs, in fact for all chronic coughers, losers of weight and tired people. We urge chest x-ray for all positive reactors to the tuberculin tests.

Your committee is glad to be of assistance in the interpretation of chest x-ray films sent to them by any member of the Society. This service is increasing, yet we urge more and more use of it.

Robert B. Kerr
Robert M. Deming
M. Dawson Tyson

On the recommendation of the Committee on Officers' Reports, the report was adopted.

Dr. Parsons, chairman of the Committee on Memorials and Communications then presented four communications for consideration. The first was from the State Board of Health and concerned a proposed survey to determine the incidence of syphilis in low-wage groups, to be undertaken with the co-operation of certain large industrial plants; some discussion followed, but no action was taken. The second was a resolution submitted by the New Hampshire Maternal Health Association, Incorporated, which read as follows:

> Whereas, The American Medical Association at its Annual Convention in June, 1937, defined birth control as an integral part of preventive medicine and adopted a four-point program to provide for scientifically tested methods of contraception and instruction in their use, the four points being: to inform physicians clearly about their medical rights; to carry on research in materials and methods for the prevention of conception; to promote thorough instruction in contraception in medical schools; and to bring all dispensaries, clinics, and similar establishments under legal licensure and medical control, therefore, be it
>
> Resolved, That the New Hampshire Medical Society endorses the stand taken by the American Medical Association and recognizes the need for medically supervised contraception as an integral part of preventive medicine.

The third was a communication from the American Medical Association concerning the hospital-construction bill (No. 1230) introduced into the United States Senate, the committee recommending that approval be extended, provided that construction be limited to that proved to be urgently

needed and that adequate maintenance be assured; this recommendation was adopted, as well as a supplementary one creating a special committee to act in an advisory capacity to the executive office of the State Board of Health on any contemplated hospital-construction project under this pending legislation.

It was voted to take off the table the motion in regard to the creation of a committee on public health; and it was then voted to create a new standing committee, the Committee on Public Health, consisting of three members. Subsequently, the tabled motion that the Committee on Public Health be instructed to consider the problem of the State Laboratory as a real competitor of the clinical pathologists was taken off the table and was approved. Furthermore, the accepted motion creating an advisory committee in regard to hospital construction was amended so that the matter was referred to the Committee on Public Health.

The final communication presented by the Committee on Memorials and Communications was a letter from the New Hampshire Farm Bureau Federation in regard to the formation of a co-operative to underwrite the costs of medical care to low-income groups in rural areas. Since accurate facts concerning the need for such a plan are lacking, the committee recommended that a special committee be appointed by the President to co-operate with representatives of the Farm Bureau in a thorough investigation of the problem. Dr. D. G. Smith, of the Committee on Officers' Reports, moved that part of the recommendation creating the appointment of a special committee be not accepted, that the rest of the recommendation be accepted and that the matter be referred to the Committee on Medical Economics, as had the question of enabling legislation covering medical-care insurance; it was so voted.

Dr. Benjamin P. Burpee then presented the report of the Committee on Infancy and Maternity.

Report of the Committee on Maternity and Infancy

This is the seventh year during which the Committee on Maternity and Infancy of the New Hampshire Medical Society has conducted a study of maternal deaths, infant deaths and stillbirths that occurred during the preceding year. The study, as in the past, is an effort to analyze the causes of maternal deaths and to appraise, so far as possible, the problems relating to the causes of deaths among mothers and babies. All agree that, although the maternal death rates in New Hampshire and in the United States as a whole have shown an encouraging downward trend, there are still too many mothers and too many infants under one year of age who die needlessly each year.

The committee has endeavored to collect as complete data as possible on each maternal death reported, and has analyzed the facts obtained with as impartial but scrutinizing care and judgment as is possible in cases where only abstract material is considered. No absolute or arbitrary conclusions can be drawn from such a study, but one can evaluate the cold facts in each case and suggest alternative procedures that might have aided in preventing the death. All this, of course, is done with the advantage of retrospect and without the element of the personal responsibility and emergency factors experienced by the physician in actual charge of the case.

The committee's remarks and conclusions must be understood at the outset to be entirely impersonal, since the name of the physician, the name of patient, the location of death and other identifying information are never known to the committee members. Only one person acting as agent must of necessity know these facts in order to collect the necessary data. That person is the director of the Maternal and Child Health Division of the State Board of Health. The study this year, as in the past, has been carried on with the co-operation of the Division of Maternal and Child Health of the State Board of Health.

Since the annual meeting of the Society last year the committee has held five meetings. These were as follows: November 20, 1940, and February 19, March 5, April 16 and April 28, 1941.

At each meeting the committee studied carefully the information collected on each case and considered every phase of the problem. Materials used in gathering data consisted of a copy of the death certificate, answers to questionnaires sent to the physician who signed the death certificate, and in most cases, facts gathered by personal interview of the agent with the physician and data from the hospital record. All cases were studied by number and all correspondence was transmitted through the Division of Maternal and Child Health.

After the cases were studied a letter was sent to the physician reporting the case, giving the committee's comments and whatever important suggestions the committee felt would be of interest or value. In some cases quite an interesting correspondence ensued between the physician and the committee, via the Division of Maternal and Child Health, in which frank comments were made from both sides. This seems to be a most wholesome practice, since out of such a medium of debate and study some excellent educational and helpful suggestions are bound to spring. The committee believes that much mutual benefit has been secured by this means.

Regarding the study of infant deaths under one year, the committee thought that the best that could be done was to reappraise these deaths as to causes reported. There were too many infant deaths reported to enable the committee to secure full data on all. Therefore, in the following pages a tabulation of causes will be set forth and some conclusions drawn.

Stillbirths were analyzed also from the standpoint of causes. Questionnaires were sent out to physicians reporting stillbirths, and excellent co-operation was obtained in the returns. A tabulation of chief causes will follow in the report.

Maternal Deaths

The committee has tabulated the maternal deaths for the calendar year ending December 31, 1940, according to the *International Classification for the Causes of Deaths*

(fifth revision). There were 24 maternal deaths reported, and all were studied. Following an appraisal of each case, the committee then reclassified the deaths into categories of causes according to the data received and conclusions drawn from that data, as follows:

Code No.	Cause of Death		No. of Deaths
140	Abortion with mention of infection		3
142 (*b*)	Ectopic gestation (without mention of septic conditions)		1
146 (*c*)	Post-partum hemorrhage		4
147 (*b*)	Puerperal septicemia (not specified as abortion)		1
147 (*d*)	Acute pulmonary embolism		3
148 (*a*)	Puerperal eclampsia		8
148 (*b*)	Mild toxemia of pregnancy		1
149 (*b*)	Other accidents of childbirth		3
	Cesarean section	1	
	Mesenteric thrombosis	1	
	Heart failure	1	
			24

After thorough study of each case, taking into consideration the events leading up to the death of the patient, the past history and the hospital record, the committee reclassified the cases as to causes of deaths, as follows:

Cause of Death		No. of Deaths
Toxemias of pregnancy (eclampsia and mild toxemias)		8
Cesarean section, with complication (without consideration for indications)		2
Post-partum hemorrhage		3
Puerperal sepsis		1
Causes not due to pregnancy		2
Cardiac failure	2	
Accidents of pregnancy		3
Pelvic thrombosis, with embolism	2	
Ectopic pregnancy	1	
Abortions		3
Unclassified		2
		24

Cases were further classified into three categories,—Group I, those in which the patient was at fault, owing to neglect, failure to seek medical advice prenatally and so forth; Group II, those in which the obstetric treatment was inadequate; Group III, those which were apparently unavoidable,—as follows:

Classification	No. of Deaths
Group I	3
Group II	9
Group III	10
Unclassified	2
	24

In the unclassified group, one record was incomplete, and one death was not deemed to have been due to maternal causes.

The causes of death in Group I, in which the patient was at fault, were as follows:

Cause of Death	No. of Cases
Induced abortion	1
Toxemia of pregnancy (no prenatal care sought)	1
Hemorrhage (atony of uterus—patient in extremis when seen)	1
	3

The causes of death in Group II, in which the obstetric care was inadequate, were as follows:

Cause of Death	No. of Cases
Cesarean section	1
Criminal abortion	2
Eclampsia	3
Post-partum hemorrhage	2
Pelvic thrombosis with pulmonary embolism	1
	9

The causes of death in Group III, in which the deaths were apparently unavoidable, were as follows:

Cause of Death	No. of Cases
Intestinal obstruction	1
Eclampsia (cesarean done in 1 case)	4
Pelvic thrombosis	1
Pelvic abscess (furunculosis)	1
Ectopic pregnancy	1
Cardiac failure	2
	10

In comparison with the study of 1939 deaths, it is interesting to note that toxemia of pregnancy was still the chief cause of death. Only three fatal cesarean sections were performed, in contrast with five recorded for 1939. Two of these sections were deemed responsible for the patients' deaths; the other was done on a patient with marked toxemia, and the death was considered to have been due to the toxemia rather than to the operative procedure per se.

In the 1940 series, 23 cases were delivered in hospitals and 1 in the home. The following data on urban and rural residency are also of interest:

County	Urban Cases	Rural Cases	Total Cases
Belknap	2	0	2
Carroll	0	1	1
Cheshire	0	1	1
Coos	1	0	1
Grafton	1	1	2
Hillsborough	8	2	10
Merrimack	1	0	1
Rockingham	3	0	3
Strafford	1	0	1
Sullivan	2	0	2
	19	5	24

No study is of value unless it is compared with findings of previous years. There were a few noticeable improvements found this year over those of last, and some conditions remained about the same. It is worthy to note that the co-operation manifested by physicians in reporting findings, giving data and answering questionnaires was greatly improved. Of twenty-four questionnaires sent out, all but one were returned, with excellent and complete data. When visited, the physicians showed a genuine interest in the study and were most anxious to discuss all features of the case.

As indicated above, only three cesarean sections were performed, as compared with five for the previous year. Five autopsies were performed in 1940, however, as compared with six the year before. Indications for post-mortem examinations varied somewhat in the two years, since more cases were due to definite causes this year. The committee believes, however, that whenever there is a doubt of the cause of death, an autopsy should be secured, if possible.

Hospital records were more complete than in former years, and it was noted with gratification that more physicians made out their own records instead of depending on hospital nurses to do so.

There is still a great deal to be desired in reference to hospital staffs requiring consultations on all operative deliveries. There has been a noticeable improvement in hospital technic and equipment. In some hospitals, new nurseries have been installed, which are well equipped for the isolation of infants. Other hospitals have secured incubators, put on fire escapes, improved plumbing facilities and sterilizing equipment and so forth. This forward movement on the whole is encouraging and is a trend that the committee believes is due to more efficient hospital administration and better co-operation with public-health agencies.

The ever-discouraging feature of any study on maternal deaths is the fact that toxemias of pregnancy continue to lead the list of all causes listed. Particularly discouraging is the fact that even when a diagnosis of toxemia of pregnancy is obvious from the records, little seems to be known by most physicians as to the treatment. Either very inadequate and futile "stabs in the dark" are made in treatment, or else no treatment is given. Not one case of toxemia studied in 1940 could be deemed as having been treated adequately.

The committee realizes that the treatment of toxemias of pregnancy is a controversial subject. Nevertheless, this fact seems to be no excuse for lax treatment of the patient with toxemia. The committee believes that patients with mild toxemias are allowed to go too long without proper treatment. A persistent rise of blood pressure over what is known to be the patient's normal pressure before pregnancy should be considered evidence of toxemia, even in the absence of albumin in the urine. Because of the persistent problem arising out of uncertainties in the treatment of toxemias, the committee will include in its recommendations a simple outline for the treatment of toxemias (this was included in the 1938 report). Let it be understood that the committee does not set itself up as an infallible authority in this matter, but believes the suggestions may prove of value to some.

The committee has presented an analysis of maternal deaths as to causes and as to facts that might be of interest as contributing to the death of the patient. It may be well to include at this point a tabulation of maternal death rates for a series of past years in order to note the trend in maternal mortality statistics:

Year	No. of Deaths	Maternal Mortality Rate Per 1000 Live Births
1930		6.2
1932		5.4
1933		6.3
1934		5.4
1935	46	6.1
1936	37	4.8
1937	34	4.3
1938	24	3.8
1939	25	3.1
1940	24	2.8

It will be noted that, up to 1936, maternal death rates had no appreciable variation. From 1936 on, the rates show a steady downward trend. This is probably not stastistically significant as yet, owing to the smallness of the series of years. However, since New Hampshire's birth rate has remained almost constant for many years, such a downward trend, if continuous, will no doubt prove significant. The committee would like to think that this downward trend is due to better obstetrics or at least to concerted efforts on the part of the medical profession to improve technics at the time of delivery and to better prenatal care. Time alone can tell, and efforts must forever be strengthened. As yet it is not wise or logical for the profession to take much credit regarding the lowered mortality rates in maternity.

Infant Mortality

The study of causes for infant deaths under one year was conducted by the committee from data appearing on the death certificates only. The large number of infant deaths reported did not permit an exhaustive analysis. However, the committee has tabulated infant deaths by causes as they appeared on the death certificate. The ages at which the deaths occurred were separated into three periods.

It is obvious again, as in former reports, that prematurity was by far the chief factor leading to the death of the newborn. There was an improvement in the reporting of causes of deaths this year over that of 1939, fewer vague and meaningless diagnoses being recorded. Twenty-one autopsies were performed. This is a distinct improvement over last year. Following is a tabulation of causes of death in infants under one year of age divided into periods as to when the deaths occurred:

Cause of Death	No. of Deaths
Deaths occurring during the first day of life	
Prematurity	75
Congenital malformations	14
Cerebral hemorrhage	7
Birth injury	7
Asphyxia	2
Syphilis	1
Atelectasis	4
	110
Deaths occurring in the first month, exclusive of the first day	
Prematurity	21
Congenital malformations	10
Intracranial hemorrhage	7
Birth injury	5
Pneumonia	4
Gastroenteritis	3
Hemorrhagic disease of newborn	3
Whooping cough	2
Enlarged thymus	1
Unknown	1
	57
Deaths occurring from the second to the twelfth month, inclusive	
Bronchopneumonia	49
Suffocation	9
Congenital heart	7
Meningitis	7
Gastroenteritis	6
Congenital malformations	6
Malnutrition	5
Prematurity	5
Unknown	5
Accidents	4
Congenital syphilis	1
Intussusception	1
Blood dyscrasias	2
	107

The total number of infants who died in 1940 under one year of age is 274; this is 33 less than that for 1939, when 307 infants died.

The total deaths from prematurity for 1940 numbered 101. It will be seen from the table that 75 of these infants died during the first day of life. Since such an astounding number of infants are lost owing to prematurity, it may be well to outline briefly a few important considerations in the care of the prematurely born infant. The committee believes that prematurity as a cause of death is, in many cases, preventable. The mere fact that an infant is born with a birth weight of 5½ pounds or less seems little excuse for the infant's death, provided, of course, that the infant has sustained no cerebral injury, has attained viability, and is in all other respects free from deformity or abnormality. The care of the premature infant involves, therefore, the following principles:

1. Extreme care should be exercised by the physician, nurses and attendants at the time of delivery to avoid trauma, unnecessary exposure or rough handling. "Jackknifing" as a method of resuscitation is condemned. Diagnosis before the onset of labor or during labor as to the possibilities of a premature infant should be attempted, and proper planning for the delivery should be considered. Teamwork practiced by the doctor, mother and nurse is essential to afford the best outcome. Equipment for resuscitation of the infant should be available at the time of de-

livery, and means for maintaining normal body temperature should be at hand. Time is a most important factor in the care of the newborn premature infant. Most of them die during the first few hours after birth and during the first day. After the first day, the infant has an increasingly good chance for life.

2. During labor, barbiturates should be cut down to a minimum. Premature infants do not tolerate sedation, particularly by barbiturates, and many infants born prematurely cannot be resuscitated under conditions in which the mother was given the usual dosage of barbiturates.

3. The maintenance of as nearly normal body temperature as possible is essential. Avoidance of excessive temperature should also be considered. An improvised heated crib built of simple materials and designed to keep fairly constant temperature is satisfactory. Heat can be provided by the use of hot-water bottles or by a protected electric-light bulb. A thermometer should be available to check the temperature of the crib at frequent intervals. (Plans for such a heated crib can be obtained from the committee or from the Division of Maternal and Child Health of the State Board of Health on request.)

4. Careful and scrupulous attention should be paid to avoidance of infections, particularly upper respiratory infections. Attendants should wear masks at all times.

5. Extreme care should be taken to provide adequate and proper nursing care and supervision of the premature infant. The physician should supervise all phases of the care, including feeding, handling and any special treatment. Transfusions are at times necessary. Handling of the infant should be reduced to an absolute minimum, and avoidance of drafts, changes of temperature and so forth should be considered.

6. Recent and studied observations have shown the definite value of the use of vitamin K at the onset of labor. It is a well-known fact that the prothrombin concentration of any newborn's blood drops markedly almost immediately after birth. This is, in some cases, responsible for cerebral hemorrhage or bleeding. This fact is particularly of significance in the premature infant, whose blood prothrombin level and all blood mechanisms are relatively unstable. It is therefore recommended that synthetic vitamin K be given as a routine procedure during labor. Best results are obtained by giving vitamin K within two hours before birth. However, no matter when given during the latter part of labor, definite results are obtained. The dosage is recognized to be 3.2 mg. given intramuscularly. If synthetic vitamin K has not been given to the mother during labor, then it should be administered to the infant immediately after birth. The dosage is also 3.2 mg., given intramuscularly.

The committee recognizes the multiplicity of problems arising in the care of the premature infant and cannot attempt to answer all of them. The foregoing is included for what value it may prove in the handling of some future case. Two facts are certain in considering care of the premature infant: first, that special care and attention must be provided and, secondly, that time cannot be wasted in applying the best and most recognized treatment procedures.

The infant mortality rates per 1000 live births by years are as follows:

Year	Rate
1935	54
1936	47
1937	48
1938	48
1939	45
1940	32

Stillbirths

One hundred and eighty-seven stillbirths were reported in 1940, a figure that compares with 211 in 1939. Questionnaires were sent to all physicians reporting stillbirths, and answers were received for 149 cases.

The causes of stillbirths were tabulated as follows:

Cause	No. of Cases
Prematurity	29
Congenital deformity	27
Cause unknown	24
Instrumental delivery	17
Other causes (varied)	17
Toxemia of pregnancy	16
Hemorrhage (placenta previa or premature separation)	11
Prolapsed cord	4
Syphilis	3
Cerebral hemorrhage	1
	149

Twenty-nine infants were stillborn owing to prematurity, and 27 had some congenital malformation; in 1939, the corresponding figures were 46 and 15. The latter cause is of course unpredictable and unpreventable, whereas premature deliveries might be preventable in the light of recent medical practices involving careful prenatal supervision.

If one studies the foregoing tables on causes of death in infants under one year of age and on stillbirths, the total number of infants lost because of premature births was 130.

There seems to be no conclusion that can be drawn from the number of stillbirths reported during the past few years as shown by the following table:

Year	No. of Cases
1937	181
1938	242
1939	211
1940	187

Comments and Recommendations

As in the past the committee has prepared certain comments and recommendations based on the study carried out. Some definite problems have presented themselves as a result of this year's study. It will be noted that toxemias of pregnancy present a real problem, especially so far as treatment is concerned. Because of continued confusion as to the treatment of toxemia or lack of knowledge as to any type of treatment that might be tried, the committee is including in this report a repetition of the recommendations made in the 1938 report concerning the treatment of toxemia. The committee realizes that no one treatment can be set up as infallible; nevertheless, a simple routine as follows may prove effective:

Absolute quiet.
Sedation (a barbiturate).
Concentrated magnesium sulfate and glucose intravenously (the dose should be regulated according to the needs of the individual case).
Rapid digitalization in all cases with convulsions or threatened with convulsions (for the average patient, a total dose of 20 to 25 gr. within twenty-four hours).

The main principles of the treatment consist in sedation, dehydration and, when necessary, cardiac stimulation.

The committee wishes to refer to recommendations made in previous years, with particular reference to indications for cesarean section. A rather comprehensive outline of indications for sections was included in the 1940 report. Other recommendations were that laboratory facilities be used when available for aiding in the diagnosis of conditions that might lead to hazards during delivery; that the proper use of the x-ray in pelvimetry is of value when performed by a competent radiologist; that an *accouchement forcé* done for any condition whatever is condemned; that manual removal of a placenta is unjustifiable unless there is excessive and uncontrollable bleeding; and that more frequent consultations be demanded by hospital staffs for operative deliveries.

Recommendations based on the study of 1940 maternal deaths are as follows:

1. Since there have been more deaths from abortions reported for the year 1940, the committee believes that every effort should be made on the part of physicians and authorities to apprehend the offenders and take steps to put a stop to such practices. This recommendation has reference to induced abortions performed without therapeutic indications.

2. The committee considers that too few post-mortem examinations are performed. Any death the cause of which is obscure should be studied in the light of post-mortem findings. It is the belief that most requests for autopsies will be granted if physicians make an effort to secure permissions. Post-mortem vaginal examinations can always be done and may reveal the cause of death.

3. The committee suggests that, in areas where such a procedure is possible, physicians who abhor doing obstetrics and who have no interest in its practice be urged to refer such cases to an obstetrician or to a physician whose chief interest is obstetrics.

4. More care should be exercised in making diagnoses of conditions arising in the prenatal and postpartum periods and of conditions occurring at the time of delivery. Diagnoses appearing on death certificates indicate, in some cases, the failure or lack of effort in coming to definite conclusions. Laboratory facilities, x-rays and careful examinations should be utilized in all cases.

5. Since the use of the sulfanilamide group of drugs has been increased, and since its successful use is dependent on proper dosage, the committee recommends that hospitals make the technics and the tests for blood concentration of these drugs available for physicians' use. To be effective the concentration of sulfanilamide in the blood should be brought up to 10 mg. per 100 cc. and maintained at that level. If the dose prescribed does not attain this end, it is in all probability not adequate. For sulfathiazole the blood concentration should be brought to 3.0 or 3.5 mg. per 100 cc. and maintained at that level.

6. The committee wishes to repeat that there is a tendency on the part of physicians to allow toxemias to continue too long without treatment. A persistent elevation of blood pressure over the known normal for the patient should be considered indicative of toxemia, even without the appearance of albumin in the urine. It is safer to consider that a patient has a mild toxemia until proved otherwise than to ignore it. Abnormal gains in weight should always be viewed with suspicion.

7. The committee recommends that patients be continually urged to seek prenatal supervision. Proper prenatal care should include visits of at least once monthly for the first seven months, and thereafter twice monthly. More frequent visits should be advised during the last trimester if indicated. Prenatal care should include a complete history and physical examination, including a Wassermann test at the outset and follow-up blood-pressure readings, urinalyses, hemoglobin determinations and blood examinations at monthly intervals. Careful records of weight should be kept.

8. Physicians should be discouraged in attempting to perform operative deliveries in the home.

9. Attempts to perform versions without deep anesthesia should be frowned on.

10. It is recommended that physicians keep practical records on all obstetric patients for whose care they are responsible.

11. The committee believes that the physician should make out the hospital record rather than to depend on nurses to do it for him.

12. Whether a delivery is in the hospital or home, the physician should not leave until he is certain the mother's condition and that of the baby are satisfactory.

13. The committee has observed that, in several cases reported this year, sudden deaths occurred on the first day the patients were allowed out of bed. In each case there had been a rise in temperature during the puerperium. It is believed that in cases in which the temperature becomes elevated the possibility of a pelvic thrombosis should be considered and that the patient should be kept in bed until the temperature has remained normal for at least three days. Emboli are common in cases of pelvic thrombosis.

Finally, the committee is most grateful for the excellent co-operation shown this year by physicians. The returns on questionnaires were especially helpful. The frank and complete comments made on many cases were most appreciated. Continued co-operation, interest and further suggestions for this study are earnestly solicited. The committee also expresses its thanks and appreciation to the hospitals that have helped make this study possible. To the Division of Maternal and Child Health of the State Board of Health, the committee expresses its appreciation for the facilities of the division and for the work performed in helping to prepare this report.

ROBERT O. BLOOD, *Chairman*
BENJAMIN P. BURPEE
MARION FAIRFIELD

After commending the committee for its excellent work, Dr. D. G. Smith, of the Committee on Officers' Reports, recommended the adoption of the report and the mailing of a copy to each hospital and physician in the State; it was so voted.

The report of the Committee on Child Health was then presented.

Report of the Committee on Child Health

Your committee held five meetings during the year.

There was considerable discussion at our meetings of the law making compulsory the reporting of children

with impaired hearing. The State Board of Health asked our advice regarding the formulation of rules and regulations for the proper execution of the law.

The committee has continued to follow as closely as possible progress in child-health work, especially as exemplified by the American Academy of Pediatrics. We have continued in close co-operation with the Division of Maternal and Child Health.

At the suggestion of the committee, Dr. Franklin Rogers delivered a radio broadcast on the preschool child over Station WFEA. This has also appeared in *New Hampshire Health News.*

COLIN C. STEWART
TRAVIS P. BURROUGHS
FRANKLIN N. ROGERS

The report was accepted.

Dr. Henry H. Amsden read the report of the necrologist.

Report of the Necrologist

The following deaths have occurred since the last annual meeting:

NAME	ADDRESS	DATE DECEASED
Ballard, Clarence P.	Penacook	March 4, 1941
Bartlett, Walter A.	Manchester	August 12, 1940
Beaton, Alexander A.	Franklin	August 22, 1940
Burtt, Edward D.	Lincoln	December 2, 1940
Clow, Fred E.	Wolfeboro	January 4, 1941
Gilman, Louis L.	Rochester	April 11, 1940
Goldsmith, Chester L.	Winchester	July 26, 1940
Hubbard, Osmon H.	Keene	January 5, 1941
Huse, Ernest L.	Meriden	December 21, 1940
Mackenzie, Nicholas Y. B.	Andover	November 27, 1940
Marks, Homer H.	Berlin	April 11, 1941
Morrill, Sibley G.	Concord	December 28, 1940
Pherson, Frank J.	Manchester	August 27, 1940
Sheldon, Harold E.	Troy	September 24, 1940

HENRY H. AMSDEN

As matters of new business, Drs. Joseph J. Cobb, of Berlin, Alfred Daudelin, of Nashua, Frank S. Lovering, of Moultonborough, Allen P. Richmond, formerly of Dover, and Lewis G. Hildreth, of Marlboro, were made affiliate members of the Society.

Dr. Wilkins spoke in favor of a change in the by-laws whereby one dollar from the dues of each member would be contributed to the Benevolence Fund, rather than fifty cents; he made a motion to this effect, which was seconded and laid over until the following day.

Dr. D. G. Smith moved that the Advisory Committee on Medical Relief be discharged and that its duty be transferred to the Committee on Medical Economics; it was so voted. He then read a letter from the Commissioner of Public Welfare requesting the approval of Dr. L. Theodore Togus's acceptance of his appointment as a member of the Medical Advisory Committee on Eye Conditions; he moved approval, and it was so voted.

The first meeting of the House of Delegates was adjourned at 11:00 p. m.

* * *

The House of Delegates convened at the Hotel Carpenter, Manchester, on May 13, 1941, at 8:30 a.m., with Vice-Speaker Timothy F. Rock, of Nashua, presiding.

The following members answered the roll call:

The President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Norris H. Robertson, Keene
Leslie K. Sycamore, Hanover
Frederic P. Lord, Hanover (alternate for Willard A. Bates, Littleton)
Deering G. Smith, Nashua
Henry O. Smith, Hudson (alternate for Charles H. Cutler, Peterboro)
Clarence E. Dunbar, Manchester
George V. Fiske, Manchester
George C. Wilkins, Manchester (alternate for Luther A. March, Nashua)
Charles H. Parsons, Concord
Herbert B. Messinger, Franklin (alternate for Robert J. Graves, Concord)
Martha I. Boger-Shattuck, Portsmouth
Frederick S. Gray, Portsmouth
Anthony Peters, Portsmouth (alternate for James Sanders, Rye)
Edna Walck, Dover
Henry C. Sanders, Jr., Claremont

Dr. D. G. Smith, for the Committee on Officers' Reports, moved the acceptance of the first set of changes in the by-laws proposed on the previous day and the abolishment of the Committee on Maternity and Infancy; after some discussion it was so voted. He then moved the adoption of the change raising the annual contribution to the Benevolence Fund from the dues of each member from fifty cents to one dollar; it was so voted.

A letter relative to the need of more medical officers for the Reserve Corps and for active duty was discussed.

The second meeting of the House of Delegates was adjourned at 9:15 a.m.

* * *

The House of Delegates convened at the Hotel Carpenter, Manchester, on May 14, 1941, at 8:30 a.m., Vice-Speaker Rock presiding.

The following members answered the roll call:

The President, *ex-officio*
The Secretary-Treasurer, *ex-officio*
Clarence O. Coburn, Manchester (alternate for Richard W. Robinson, Laconia)
Francis J. C. Dube, Center Ossipee
Frederic P. Lord, Hanover (alternate for Willard A. Bates, Littleton)
Deering G. Smith, Nashua
Henry O. Smith, Hudson (alternate for Charles H. Cutler, Peterboro)

Clarence E. Dunbar, Manchester
George V. Fiske, Manchester
George C. Wilkins, Manchester (alternate for Luther A. March, Nashua)
Frederick S. Gray, Portsmouth
Anthony Peters, Portsmouth (alternate for James Sanders, Rye)
Edna Walck, Dover
Albert E. Barcomb, Rochester

Dr. Lord reported for the Committee on Nominations as follows:

OFFICERS

President: Charles H. Dolloff.

Vice-President: Henry C. Sanders, Jr.

Secretary-Treasurer: Carleton R. Metcalf.

Councilors (five-year term): Cleon W. Colby, John A. Hunter.

Trustee (three-year term): Samuel T. Ladd.

House of Delegates: speaker, Timothy F. Rock; vice-speaker, Charles H. Parsons.

Delegate (American Medical Association): Deering G. Smith.

Alternate Delegate (American Medical Association): Emery M. Fitch.

Necrologist: Henry H. Amsden.

STANDING COMMITTEES

Amendments to Constitution and By-Laws: James B. Woodman, Frederick S. Gray, W. J. Paul Dye.

Child Health: Colin C. Stewart, Jr., Travis P. Burroughs, Franklin N. Rogers.

Control of Cancer: George C. Wilkins, Howard N. Kingsford, George F. Dwinell.

Maternity and Infancy: Robert O. Blood, Benjamin P. Burpee, Marion Fairfield.

Medical Economics (three-year term): Richard W. Robinson.

Medical Education and Hospitals: John P. Bowler, James W. Jameson, Herbert L. Taylor.

Mental and Social Hygiene: Charles H. Dolloff, Benjamin W. Baker, John B. McKenna.

Public Health: Chester L. Smart, Harris E. Powers, Anthony E. Peters.

Public Relations: Charles H. Dolloff, Henry C. Sanders, Jr., Carleton R. Metcalf, Robert J. Graves, Joseph N. Friborg.

Publication: Carleton R. Metcalf, Warren H. Butterfield, Ellsworth M. Tracy.

Scientific Work: Carleton R. Metcalf, Frederick P. Scribner, Nathan T. Milliken.

Tuberculosis: Robert B. Kerr, Robert M. Deming, M. Dawson Tyson.

The report was accepted. The Secretary was instructed to cast one vote for Dr. Dolloff as president; this was done, and he was declared duly elected.

Dr. J. J. Morin, of Rochester, and Dr. H. L. Taylor, of Portsmouth, were nominated for the office of vice-president, in addition to Dr. H. C. Sanders, Jr. Following written ballots, it was announced that Dr. Sanders had received 13 votes, and Dr. Morin, 1; Dr. Sanders was declared duly elected.

The Secretary was instructed to cast one ballot for Dr. Metcalf as secretary-treasurer, and one for the remainder of the officers and committee members as nominated; this was done, and all were declared duly elected.

Dr. Coburn moved that the Society, through the House of Delegates, extend a vote of thanks to Dr. H. O. Smith for his work in bringing the history of the New Hampshire Medical Society to date and compliment him on his efforts as chairman of the committee in charge of the one hundred and fiftieth anniversary; it was so voted. He then moved that a vote of appreciation and thanks be extended to the committee that had had charge of arranging the very interesting and popular medical exhibit; it was so voted.

Dr. Metcalf moved that a vote of gratitude be extended to the members in Manchester who had arranged the details of the meeting; it was so voted.

Dr. Wilkins raised the question whether certain moneys from the General Fund should be transferred to the Benevolence Fund, thus providing a small income that could be used for needy cases. After some discussion, it was voted that the trustees be given power to expend money from the General Fund for the relief of needy members.

After some discussion, it was the consensus that a social gathering similar to the one held on the previous evening be held for the next two or three years.

It was further decided that the 1942 meeting be held in Manchester.

Dr. Wilkins moved that a vote of thanks be extended to Dr. George Dwinell for his work in scheduling and arranging the commercial exhibits during the previous six or seven years; it was so voted.

Dr. H. O. Smith called attention to the fact that the badge being used by the members of the Society was a replica of one presented to the members in 1906 by the president, Dr. F. A. Stillings;

at that time it was voted that the button be made a permanent badge, but the vote was forgotten three years later. He also said that at one time the Society had had a valuable reference library, its nucleus being the gifts of Dr. Joshua Brackett and his widow. In 1891, there were over three hundred volumes at Dr. Irving A. Watson's office at the State House; however, no trace of these books can be found.

The third and final meeting of the House of Delegates was adjourned at 9:25 a.m.

CARLETON R. METCALF, *Secretary.*

MEDICAL PROGRESS

THE TREATMENT OF DEHYDRATION IN PATIENTS WITH HYPERTROPHY OF THE PROSTATE, RETENTION OF URINE AND IMPAIRMENT OF RENAL FUNCTION*

F. A. SIMEONE, M.D.†

BOSTON

OF recent years, the problem presented by hypertrophy of the prostate and the interference with the normal function of the urinary organs caused thereby has been discussed in the literature largely by articles dealing with the technic of prostatectomy, whether by the transurethral, transvesical or perineal routes, and with analyses of end results of such operations. Discussions of measures to improve the badly disorganized general condition shown by many such patients, so that they may survive any form of operation, have been less numerous. It is nevertheless essential to point out that the gratifying reduction of both mortality and morbidity of earlier years was the result of improvements in the preoperative and postoperative conduct of the patients rather than of improvements in operative technic, however valuable these may have been. The removal of the prostate in two stages resulted from a realization of the necessity for restoring the patient's disordered metabolic and chemical processes to a condition as nearly normal as possible before subjecting him to prostatectomy. Thus, in 1914, Pilcher[1] stated:

> The chief indication is primarily to relieve the retention of urine rather than the removal of the prostate. . . . If we could dissuade surgeons, as a rule, from the course of immediately taking out a prostate which is known to be obstructive, we would do much to help the cause of the prostatics.

The cardiovascular and the renal excretory systems bear the brunt of the secondary effects of prostatic hypertrophy. In patients of the age group in which hypertrophy of the prostate occurs, the heart and circulation are often already the seat of natural changes that decrease, sometimes practically to annihilation, the reserve that these systems possess in men of younger age groups. When obstruction to the outflow of urine from the kidneys occurs, the burden of an increased arterial blood pressure is added to the already overtaxed heart. When such elevation of blood pressure has existed for a long time, a rapid fall of blood pressure following decompression of the urinary passages, such as that afforded by catheterization, may induce profound changes in the blood flow through vital organs. Certain cases of disorientation, coronary occlusion and oliguria or anuria after too rapid decompression of the obstructed bladder may be referable to such circulatory changes. The treatment of the failing heart and circulation thus assumes a major role in the management of patients with prostatic hypertrophy of long standing.

The secondary effects of the obstructing prostate on the renal excretory system itself are referable principally to infection and to derangement of renal function. The burden that infection places on the bodily economy needs no emphasis. The service that chemotherapy, particularly the use of sulfanilamide and sulfathiazole,[2, 3] has rendered to those suffering from infection in the urinary tract is now generally recognized. The fact that these drugs act in especially small

All articles in this series will be published in book form; the current volume is *Medical Progress: Annual, 1940* (Springfield, Illinois: Charles C Thomas Company, 1941. $4.00).

*From the Urological Clinic, Peter Bent Brigham Hospital.

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amounts in urinary infection is particularly fortunate. To be sure, they are not curative in the presence of obstructing lesions of the urinary tract, but this does not detract from their value as adjuncts to bladder drainage in preparing patients with urinary retention and infection for prostatectomy.

Among the widespread effects of renal insufficiency accompanying prolonged urinary obstruction, the most serious from the point of view of the urologic surgeon is the derangement in water balance and in the electrolyte pattern of the body fluids. Clinically, interest centers on the treatment of the dehydration that almost invariably attends the deranged electrolyte composition of the body fluids. The importance of water and electrolytes for the proper execution of body functions cannot be overemphasized. The maintenance of normal metabolic reactions, as well as such complex functions as wound healing, depends on a proper complement of water and electrolytes. A brief summary of the problems encountered in the management of water balance or dehydration in urologic patients is therefore profitable.

The proper management of dehydration depends on three premises: recognition of the state of dehydration, detection of the underlying causes and familiarity with methods available for correcting the condition.

The Diagnosis of Dehydration

The clinical signs of dehydration are easy to recognize. The skin is warm, dry, wrinkled and inelastic. The eyeballs are soft. The tongue is dry and difficult of motor control so that speech is thick. If dehydration is sufficiently severe, acetone may be detected on the breath. The sensorium is dulled in varying degrees up to coma. The pulse is rapid, but the arterial blood pressure may be normal until the condition is far advanced, when a state resembling secondary shock supervenes. Acidosis is usually present, with its attendant respiratory changes, but there may rarely be alkalosis, even when ketone bodies are being excreted in the urine.

Such clinical signs as have been described, however, usually accompany a loss of 6 per cent or more of the body weight in water. Slight degrees of dehydration are often difficult to recognize. To detect dehydration before too serious metabolic disturbances have been created, several laboratory tests have been employed. The commoner ones are the following.

Red-cell count and hematocrit determination. An increase in the number of red cells in the blood stream, as determined by red-cell counts and hematocrit determinations, has been interpreted as indicating a decreased content of water in the blood (anhydremia) and, indirectly, a decrease in the volume of interstitial fluid (dehydration). Such an interpretation is justified in the absence of primary or secondary polycythemia from other causes, such as cardiopulmonary disease. The findings, however, are not always dependable. The tests may be normal when the dehydration is slight, because of the efficiency of the homeostatic mechanisms in maintaining constancy in the blood by drawing on the fluid stores in the interstitial (subcutaneous) tissues. When dehydration has been severe and of long standing, anemia develops secondary to destruction, as well as to depressed formation, of red blood cells. A normal or subnormal red-cell count and hematocrit determination may thus be misleading.

Specific gravity of the blood and serum-protein concentration. These tests are as easily done as the red-cell count and the hematocrit determination.[4, 5] They are subject to the same faults, however. Considerable interstitial fluid may be lost before any marked change becomes apparent in the concentration of the serum or plasma protein. On the other hand, one may encounter normal plasma-protein concentrations or actual hypoproteinemia in severe chronic dehydration because of the destruction of blood proteins when anhydremia is prolonged.

Study of the electrolyte pattern of the blood plasma. Here again the numerous reactions that maintain stability within the circulating fluids militate against early detection of dehydration until the condition has advanced so far that the homeostatic mechanisms begin to fail. At this point, one almost always finds a depression in the total-base component of the blood plasma, represented largely by sodium. The base depletion, by conditioning an equivalent loss of water, may be a primary cause for the dehydration (as in patients with biliary, pancreatic and intestinal fistulas), or it may be a secondary effect of prolonged or severe dehydration that has caused an impairment of renal function. In fact, so long as there is normal renal function, — or perhaps more accurately, so long as there is opportunity for normal renal function, — the total base of the blood plasma remains normal, even in the presence of moderately severe dehydration.

The value of estimating the electrolyte pattern of the blood lies in the information derived therefrom concerning the *degree* of derangement present and the approach suggested thereby for fluid-replacement therapy.

Estimation of the fluid need of interstitial tissue. Hopps and Christopher[6] adapted the intradermal wheal-absorption test of Aldrich and McClure to the study of dehydration in surgical patients. The test consists in the intradermal injection of 0.2 cc. of physiologic saline solution to raise a wheal, whose rate of disappearance is then measured. Normally, such a wheal disappears in fifty-five or more minutes. A decreased disappearance time indicates a scarcity of subcutaneous fluid, or dehydration. Actually, however, the test probably reflects the existing osmotic conditions in the skin and subcutaneous tissues and not necessarily the lack of body water. Thus Kunde[7] found that the disappearance time decreased in cases of hydropigenous nephritis. In fact, it was possible to detect a decrease in the disappearance time even before the appearance of edema. Cases exhibiting edema, however, may still present the picture of dehydration in the broad sense of the term. Cases in which the total content of body water is normal or actually greater than normal might more properly be considered to represent disordered distribution of body water rather than dehydration.

It becomes apparent that slight changes in the concentration of plasma constituents, which may be sufficient to call into action the numerous regulatory mechanisms that preserve constancy while dehydration is progressing, cannot be detected by the usual analytical methods. Determinations of blood volume are not applicable to the study and treatment of early clinical dehydration. Evaluation of the Aldrich–McClure intradermal wheal-absorption test must wait until more experience has been had with the test. The activity of the salivary glands, however, is a sensitive indicator of the state of hydration of the body. Long before ionic changes are demonstrable in the blood, the withdrawal of interstitial fluid causes a diminution in the blood flow through the salivary glands, the secretions from which are thereupon impoverished. Thus, the best index of early dehydration is the old clinical observation of dryness of the buccal and oral mucous membranes. Dysphagia is frequently present and may appear early in patients suffering from prostatism. Since dehydration may actually be secondary to hyposthenuria, the excretion of normal, or greater than normal, volumes of dilute urine is not inconsistent with dehydration.

The value of blood chemical determinations is not to be belittled, however: they at least demonstrate the degree of dehydration, and the nature of the shift in electrolyte pattern serves as a guide for replacement therapy.

Etiologic Factors in Dehydration

For convenience of description, the causes of dehydration fall into two major groups: those in which deficiency of fluid in the body results either from lack of fluid intake or from loss of water and salts through wastage of body secretions; and those in which water is lost secondarily because of disturbances in the electrolyte composition of body fluids due to abnormal function of the kidneys. Dehydration secondary to hemorrhage or loss of plasma through denuded skin surfaces, as in severe burns, is a special case that belongs to the first category. The treatment of the dehydration encountered in the first group has been discussed in several publications (Maes and Davis[8]). The second group of cases, which are of special interest to the urologist, however, have received little attention. The frequency with which patients with urinary retention and impaired renal function exhibit a dehydration that is practically always secondary to a multiplicity of factors warrants further analysis of the nature of dehydration in patients with hypertrophy of the prostate, urinary retention and renal insufficiency.

The principal factors determining dehydration in such urologic patients are insufficient fluid intake, loss of water and salts from the body and electrolyte imbalance due to impairment of renal function, with consequent loss of body water.

Insufficient fluid intake. Metabolic activity involves a daily inevitable loss of water from the body. The greatest volume is lost by vaporization from the skin and lungs. The actual amount lost naturally varies with body temperature, muscular activity and the temperature and humidity of the environment. On the average, however, 2000 cc. a day are lost by vaporization. Next in amount is the water lost through the kidneys in the form of urine. So long as renal function is normal, the water excreted by this route varies inversely with the amount of water lost through other channels. When the water stores in the body are low, the normal kidney conserves water by excreting a concentrated urine. The diseased kidney, however, is unable to extract water efficiently from the glomerular filtrate, and water is wasted. Thus, the normal kidney can excrete an average of 35 gm.

of solids in little more than 400 cc. of urine. The diseased kidney, which is unable to concentrate beyond a specific gravity of 1.010, requires 1500 cc. to excrete the same amount of solids. Normally, the water lost in the stools is negligible.

For metabolism to proceed normally, this inevitable daily loss of body water, amounting to about 3500 cc., must be replaced, and this is accomplished by the ingestion of water with food or drink regulated by the sensation of thirst. The metabolic water derived from the breakdown and transformation of food elements seldom amounts to more than 500 cc., and in patients who have limited their food intake because of illness, water derived from this source is negligible. It is noteworthy that patients with urinary retention, renal insufficiency and nitrogen retention often are not thirsty, even though they present obvious clinical signs of dehydration. They sometimes actually complain that water "tastes bad" and refuse to drink. A vicious circle is thus established whereby dehydration, which may have begun by electrolyte derangement, is aggravated by inadequate fluid intake.

Loss of body fluid through abnormal channels. In addition to loss of water in the urine and by vaporization, which, except in degree, does not differ from the normal, fluid may be lost by various abnormal means. These are common knowledge, and the problems presented by such avenues of fluid loss as biliary and intestinal fistulas are not germane to the consideration of dehydration in the prostatic patient with renal insufficiency and nitrogen retention. The urologist is often confronted, however, with the necessity of replacing fluid lost by vomiting and sometimes by diarrhea. It should be pointed out here that vomiting in such patients is not prone to cause alkalosis but rather dehydration and acidosis because of the relative achlorhydria in azotemia. What chloride escapes in the vomitus does so in company with sodium.

Loss of body fluid because of changes in the electrolyte pattern of the blood secondary to impairment of renal function. Changes in the electrolyte composition of the blood associated with impaired renal function are varied. Irrespective of the type of renal disease, however, the principal changes are referable to the total base (principally sodium) and the chloride and bicarbonate acid ions.

The determination of the total-base equivalence of blood plasma offers an accurate index of its osmotic characteristics, which depend in large measure on the electrolyte content of the plasma. The total-base concentration in the plasma of patients with renal insufficiency is almost invariably low. The mechanism whereby salt is spared by the kidney is lost. Water is lost along with the base, and dehydration ensues. It is only with far advanced total-base depletion and disordered renal function that water is retained in spite of low total-base concentration in the blood and tissue fluids.

The mechanism for base depletion in renal disease is not entirely clear, but an important role is no doubt played by the inability of the diseased kidney to synthesize ammonia, with which to excrete nonvolatile acids as ammonium salts. Instead, they are excreted as salts of fixed base derived from the blood plasma.

In renal disease, the concentration of the chloride ion in the blood is very variable. Thus in 148 cases of varied etiology, Peters, Wakeman, Eisenman and Lee[9] found that the chloride determinations were below normal limits in 56, and above in 44. A low blood-chloride concentration, however, does indicate almost invariably a low total-base or total-electrolyte concentration in the blood, with consequent loss of water in the attempt of adjustive mechanisms to restore osmotic equilibrium. A low total base, however, does not necessarily imply a low chloride concentration. The usually reciprocal relation between the concentrations of the chloride and bicarbonate ions prevents the loss of chloride when the total base concentration has become low, that is, the bicarbonate ion variation is the principal mechanism whereby a balance is maintained between the acid and base equivalence of the blood electrolytes. Hence, acidosis usually accompanies dehydration, and base is lost from the blood without a corresponding loss of chloride.

Even in dehydration secondary to electrolyte imbalance, the very causation of the dehydration makes impossible its early detection by chemical study of the blood plasma. Changes in the electrolyte pattern of the blood are detectable by gross methods only when the causative factors have progressed to such degree that compensation for them by the usual homeostatic mechanisms has begun to fail.

Treatment of Dehydration

In the management of the postoperative patient with urinary retention and impairment of renal function, the administration of 3500 cc. of fluid every twenty-four hours is sufficient to replace the inevitable fluid loss from the body during convalescence. It ensures the availability of about 1500 cc. of water for excretion in the urine, and this is sufficient for the excretion of 35 gm. of solids even by kidneys that cannot concentrate beyond

a specific gravity of 1.010. Fluid lost through vomitus or other abnormal channels must be measured and replaced in the form of physiologic saline and dextrose solution, in addition to the 3500 cc. When the function of the kidneys is normal, it makes little difference in what form the inevitable fluid loss from the body is replaced, provided that 8 to 10 gm. of salt per day is furnished. Fluid lost through acute hemorrhage is, of course, best replaced by whole blood, and the protein-rich fluid lost in exudates, by blood plasma or serum.

The treatment of dehydration in the presence of primary or secondary electrolyte imbalance requires special consideration. The choice of the various types of fluids available for parenteral therapy depends on the nature and degree of the electrolyte imbalance, and the success of therapy depends in part on the choice of proper fluids and in part on the degree to which renal function can be restored. It is essential to remember that each case of dehydration with disturbed blood-electrolyte composition presents problems peculiar to itself, and that treatment must therefore be individualized. Nevertheless, certain general principles may be formulated.

Cases in which dehydration is mild and renal function is normal. These cases are seldom of any concern. The dehydration is usually secondary to insufficient fluid intake because of the associated illness. Fluid loss through abnormal channels plays only a small part, if any. Therapy is simple. According to the degree of dehydration, as estimated clinically, 1000 to 2000 cc. of fluid are administered parenterally, in addition to the 3500-cc. requirement for the twenty-four hours.

Cases in which dehydration is severe but renal function is adequate. These patients have experienced water deprivation for longer periods or have lost large quantities of fluid by vomitus, diarrhea or fistulas. The principles of parenteral fluid therapy are the same as those for the preceding group, but larger volumes of fluid are required. Such patients may have lost as much as 6 per cent or more of their body weight in water, and this must be replaced in addition to the basic requirement of 3500 cc. It is not uncommon, therefore, for a patient who is severely dehydrated to require 7000 cc. or more of fluid in twenty-four hours. Most of this may be given in the form of 5 per cent dextrose in distilled water, but enough of it must be in the form of physiologic saline to provide a daily minimum of 8 or 10 gm. of sodium chloride and to replace fluid lost through vomiting and diarrhea, volume for volume.

Acute severe dehydration of short duration is accompanied by the signs of hemoconcentration described above. In such cases, care must be taken to avoid too rapid administration of fluid to obviate overloading the heart by sudden dilution of the blood, which might otherwise ensue. It may thus require as long as ten or twelve hours to administer the full complement of fluid for twenty-four hours in a severely dehydrated patient. It must be emphasized that when dehydration has been of long standing, or when for other reasons the anhydremia is accompanied by loss of plasma protein and red blood cells, the blood volume is to be restored, not by means of solutions of crystalloids, but rather by means of whole blood or blood plasma. In such cases, the administration of crystalloid solutions often initiates hypoproteinemia, not only by dilution of the blood but also by actual loss of protein from the capillary bed.

Cases in which dehydration and impaired renal function coexist. Severe dehydration is known to have profound effects on both the structure and the function of the kidneys. Thus, red blood cells, casts and albumin are not uncommonly found in the urinary sediment of such patients. In addition, there is often nitrogen retention of considerable magnitude in the blood. Yet, when the renal effects are secondary to dehydration resulting from other causes, the treatment is hardly more difficult than that described for the last group above. Renal function is usually promptly restored by the administration of fluid in the form of dextrose solution. This has special value in combating the ketosis and acidosis that such patients often exhibit. Not only are water and carbohydrate thereby supplied to restore to normal the metabolism of the ketogenic substances, but proteins are spared from catabolism so that the necessity for eliminating nitrogenous products and nonvolatile acids is decreased.

The cases of dehydration due to or complicated by chronic disease of the kidneys are the most difficult to treat. It is in this group that patients with hypertrophy of the prostate and urinary retention fall. Study of these cases makes one realize the important work the kidneys perform in regulating the pattern of osmotically active ions in the blood.

The first consideration in the treatment of such patients is restoration of the blood volume to as nearly normal as possible. Unlike the cases in the two groups described above, these patients usually show no laboratory evidence of hemoconcentration. The red-cell count, the hematocrit and the specific gravity of the blood may be quite normal. Yet the skin and mucous membranes are dry, there may be acetone on the breath and in the urine, and study of the blood electrolytes may show base depletion and acidosis. While prepara-

tions are being made for relieving the urinary obstruction, therefore, these patients should receive 500 cc. or more of blood plasma, or of whole blood if there is anemia or if plasma is not available. Further restoration of fluid balance is accomplished by the administration of crystalloid solutions, and the choice of fluid depends in large measure on the characteristics of the blood electrolyte disturbance.

In contrast to the almost invariable finding of a lowered total-base concentration in the plasma of patients with dehydration and renal insufficiency, the concentration of the chloride ions may be increased to well above its normal value. This, of course, implies a drop in the concentration of the bicarbonate ions and a lowering of the alkali reserve. After the blood volume has been restored by transfusion, the next step is to induce a diuresis by the slow injection of glucose solution (1000 cc.) followed by glucose in physiologic saline solution. These solutions are alternated until the total desired volume for the twenty-four-hour period has been attained. The objection to using dextrose solution alone is that the very production of diuresis in hyposthenuric kidneys causes salt wastage and further depletion of base. Furthermore, it appears impossible for such kidneys to excrete chloride and retain base. Acidosis thus persists in spite of saline infusions, and when the diminution in alkali reserve has become sufficiently severe, these form the ideal cases for the administration of sodium bicarbonate or of sodium lactate (Hartmann–Senn solution) intravenously.

Not infrequently, the depletion of total base is accompanied by loss of chloride. When the chloride concentration of the blood is sufficiently low, the bicarbonate concentration may be quite normal. The mechanism of the decreased blood chloride is often difficult to explain. Even when there has been considerable vomiting, the cause for the hypochloremia remains obscure, since patients with even slight nitrogen retention in the blood are apt to have moderate to complete achlorhydria. As in the cases with chloride retention, the first indication is to restore the blood volume by means of whole-blood or plasma transfusion. The electrolyte concentration can then be brought to normal by the administration of glucose in physiologic saline solution. Some advocate the administration of Ringer's solution in place of sodium chloride solution. The advantages of the more complex fluid are only slight. It is in this group of cases that, owing to renal insufficiency, it is easily possible to effect a higher than normal electrolyte concentration in the blood, with consequent plethora and edema. Blood-chloride concentrations should therefore be checked at frequent intervals. The unregulated use of dextrose solution, however, can lead to equally distressing complications. The diuresis incident thereto washes base from the blood and leads to further depletion, and in addition there is actual loss of protein from the blood stream.

These urologic problems in dehydration are frequently complicated further by heart failure and peripheral edema. The same general principles outlined above still hold. The choice of fluids is based on the same principles, but the administration should be slower and the total volume of fluid should be curtailed until cardiac compensation has been restored. When diuresis begins, however, as a result of cardiac improvement due to bedrest or digitalis, fluids should be urged rather than restricted, to avoid the concentration of waste products in the tissue fluid and blood (uremia), which may result when the kidneys are unable to concentrate urine beyond the specific gravity of 1.010.

The preceding discussion has implied that fluid replacement therapy is accomplished entirely by vein. But fluid may be administered subcutaneously as well as intravenously when there is no peripheral edema. It is customary to use 4 rather than 5 per cent solutions of dextrose for subcutaneous injection. The injection of more than 2000 to 2500 cc. a day by the subcutaneous route, however, is seldom necessary. When co-operation is possible on the part of the patient, the fluid intake by mouth may be supplemented by the rectal administration of tap water, glucose solution or physiologic saline solution.

Finally, it should be emphasized that the best method of administering fluid is to encourage the patient to take it by mouth whenever possible. To overload the stomach in an effort to avoid parenteral administration of fluid, however, is a sad commentary on clinical judgment.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27341

PRESENTATION OF CASE

A thirty-four-year-old salesman entered the hospital for study.

Fifteen years before admission, the patient received a blow in his left back and felt something snap in the region of his left scapula. The part was painful on deep breathing, sneezing and coughing for three weeks, and sometime later he noticed a lack of sensation of his left hand when he dipped it in hot or cold water, at a temperature painful to his right hand. In addition an inconsistent, nonradiating, burning pain occasionally appeared in the left anterior or posterior chest, and in the left upper or lower arm. This sensation occurred at irregular intervals, lasted seconds to minutes, and varied in location. The patient visited the Out Patient Department, where a loss in tendon jerks and a decrease in pain and temperature sense were noted in the left arm. He was studied thoroughly, and since no organic cause was found to account for the condition, it was considered to be hysterical in origin. Over the fifteen years prior to admission, there was no appreciable change in the patient's symptoms, although pain sensation returned in some degree to his left hand. Immediately before entry, the patient was subjected to a routine examination for employment, and a mediastinal mass was demonstrated by x-ray film. At no time had there been cough, hemoptysis or weight loss.

Physical examination was entirely negative except for diminished pin and temperature sensations over the left arm and hand; this was too vague in its distribution to be mapped out. All tendon reflexes were absent in the left arm, but present in the right.

The temperature, pulse and respirations were normal.

Examination of the urine and blood was negative. A blood Hinton reaction was negative. Findings on lumbar puncture were normal, and a spinal-fluid Wassermann test was negative.

X-ray films of the chest showed a soft-tissue mass, 7.5 by 6.5 cm., at the posterior aspect of the left chest close to the fifth, sixth and seventh ribs. The fifth rib showed a pressure defect on its lower border, and the sixth rib was flattened and widened in the region of the mass, the changes in the flattened portion being interpreted as an old, healed fracture. The rest of the chest showed no abnormalities.

An operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. THEODORE L. BADGER:* May we see the x-ray films?

DR. AUBREY O. HAMPTON: The lateral views are not here. I think we can accept the interpretation of the size and location of the soft-tissue mass. You can see the upward concavity of the fifth rib, which looks as though it had been pushed up by pressure, and the enlargement of the sixth, with definite alteration in structure; there is no

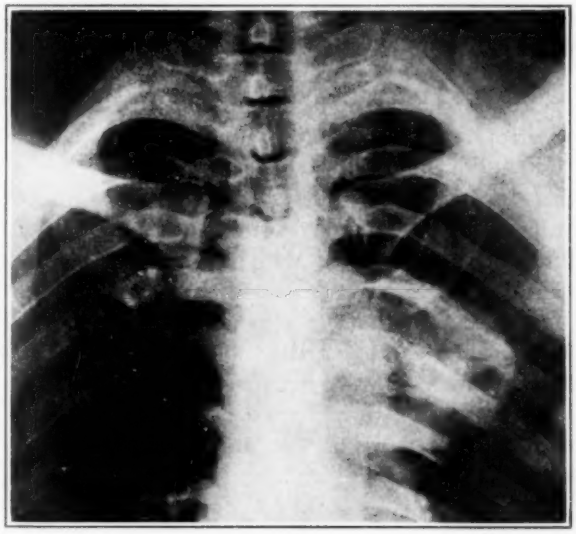

FIGURE 1.

deformity of the seventh (Fig. 1). Within this mass, there are flocculent areas of calcification, not mentioned in the report.

DR. BADGER: There is no evidence of bone destruction so far as the vertebra is concerned?

DR. HAMPTON: I do not believe so, but the vertebras are not well seen. The mass is close to the spine. One might expect it to be eroded by pressure just as this rib is. I might say that this appearance in the rib is not by any means typical of a healed fracture.

DR. BADGER: Summarizing the case briefly, we have a man who, following a blow in the left back fifteen years before admission, developed secondary symptoms in the left arm, which have recurred from time to time up to the present, essentially without change in character. In the present illness, these symptoms were fleeting pains that

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came and went in the left arm, loss of tendon jerks, and loss of sensation to cold and heat, symptoms that are almost identical to those noted fifteen years previously. At that time, no definite cause for the neurologic findings was discovered.

Do you know whether an x-ray film was taken at the time of the blow?

DR. RALPH ADAMS: No x-ray film of the chest was taken.

DR. BADGER: The important features really boil down to what was apparently a fracture of the left sixth rib when the patient received the injury. He had pleuritic pain on cough, sneezing and deep breathing, consistent with fracture of a rib. We do not know the severity of the blow. The symptoms and physical findings have not changed in any way. The neurologic findings, which were extremely vague, were the same at the time of the injury as they were at admission. The areas with loss of pin sensation are not accurately demonstrated. No mention is made either of atrophy of the hand or of differences in the two pupils, and Horner's syndrome was apparently not present. Therefore, I am at a loss to know whether this left-arm syndrome is definitely to be connected with the original trouble. Clinically, the patient was well, with no symptoms except in the left arm and hand. He was being examined for a new job. The laboratory findings were entirely negative. Had it not been for the x-ray picture, I gather there would have been no discovery of the underlying pathologic lesion.

This mass shows some calcification in its center, the left sixth rib is definitely abnormal, and there is a pressure distortion of the fifth rib posteriorly. The differential diagnosis in this case revolves around the character of the tumor mass. It seems unlikely that the tumor was malignant, because it would be unusual for a malignant tumor to be present fifteen years without more significant changes, although we might find malignant changes developing late in a primary benign tumor.

But if it was a benign tumor, how are we going to account for it? It occurs to us at the start that a severe blow in the back with considerable pain and with symptoms indicative of fracture might have had something to do with it. We wonder whether this was an unusual hematoma resulting from the fracture, which had never been observed and which had calcified over a period of years without significant change in size. That would to some extent account for the picture presented. There appears to be no definite evidence of an aneurysm. The signs are not consistent with it. The arch and descending aorta were normal, and the spinal-fluid Wassermann test was negative. Dermoids and teratomas are more likely to be seen in the anterior chest, and I believe they can be ruled out. Lymphosarcoma or Hodgkin's disease was probably not responsible.

If the mass was in any way associated with the symptoms, which persisted for years, we find ourselves reduced to three possibilities. If the mass was associated with the original injury, it should be a benign tumor — either a hematoma or a neoplasm of slow growth. Chondromas are likely to be posterior tumors in the upper portion of the chest and might be related to the bone injury. The x-ray findings were not quite characteristic of chondroma, but I think it could have been that, and the pressure symptoms for a long period of time might in part have been due to such a tumor. We must also bear in mind the possibility of neurofibroma. So far as benign tumors are concerned, neurofibroma is probably a good bet for explaining symptoms over a long period — an entirely benign tumor presenting no symptoms except neurologic ones, explainable on the basis of neurofibromatous involvement of some portion of the nerve roots. If the tumor involved the first, second or third rib, I should be more inclined to account for the neurologic symptoms, on the basis of pressure or invasion, than I am able to in the location where it actually occurred. There appeared to be no involvement of the sympathetic chain, for there is no comment about alterations in the left pupil. Also, there was no apparent atrophy of the left hand. In fact, I am a little confused about the neurologic findings: they were constant so long and yet produced no secondary changes. I am, therefore, inclined to divorce the neurologic findings from the underlying lesion in the chest and to believe that they may be explained on some other basis.

I think that this is a benign tumor, and I name chondroma, calcified hematoma and neurofibroma, in that order, as the most likely possibilities.

DR. DONALD S. KING: I saw this patient once in the Thoracic Clinic. We put neurofibroma first, with secondary changes in the ribs from pressure. Our opinion had been changed at the time the surgeons operated, but that was our original impression.

DR. CHARLES S. KUBIK: Have we ever had a neurofibroma with calcification?

DR. HAMPTON: We have had a ganglioneuroma, with calcification and secondary involvement of the ribs, but not like this. This rib should have

given us more pause. The X-ray Department made a diagnosis of neurofibroma. I do not know whether they changed later or not.

Dr. Wyman Richardson: Do you have to think of vascular tumors — hemangioma and so forth?

Dr. Hampton: The widened rib and the change in structure should make one suspect a primary bone tumor. It could be a vascular tumor, or an osteochondroma; and we have a few cases of bone changes similar to that in vascular neurofibromas. I do not know how content we should be with any diagnosis, but I think Dr. Badger's reasoning is a little better than ours.

Dr. Edward D. Churchill: I think we were a little hasty in attributing the changes in the ribs to the pressure of a neurofibroma. A neurofibroma arising from the intercostal nerves gives extreme deformity of the ribs. They look nothing at all like these ribs. They are thin, keel-like structures, completely surrounded by slowly growing tumor. All of us who saw this man made a provisional diagnosis of neurofibroma. I wish Dr. Kubik would tell us just how this tumor is destroying the tendon reflexes in the left hand.

Dr. Kubik: I cannot explain it any more than Dr. Badger could. The tumor is not in the right place to do so, and perhaps one cannot assume it was present for fifteen years, or from the time that the patient first observed neurologic symptoms. It obviously could not have been hysteria if the tendon reflexes were gone. The findings suggest more than one lesion, and therefore favor neurofibroma. There may be another lesion, possibly intraspinal, although the lumbar-puncture and spinal-fluid findings do not suggest that, in fact are rather against it.

Dr. Hampton: Osteochondromas are also multiple.

Dr. Kubik: I should not have thought of neurofibroma, because of the calcification, which I cannot remember having seen with neurofibroma.

Dr. Adams: Dr. King's note from the Thoracic Clinic reads, "The x-ray findings are characteristic of neurofibroma with rib changes, but no erosion; Dr. Churchill strongly inclines toward surgery." At operation, the sixth rib was densely adherent posteriorly and grossly deformed by intrinsic tumor. The rest of the tumor was found lying in the costovertebral gutter, with the left sympathetic chain forced backward over its posterior aspect and closely adherent. The tumor stopped abruptly at the head of the rib. The pleura could be separated from its attachments without profuse bleeding. The tumor seemed to be composed of irregular bony fragments, which felt like a bag of gravel. I thought at that time that the preoperative diagnosis of neurofibroma should be changed to chondroma. The tumor was easily removed, and the patient made an uneventful recovery.

Clinical Diagnosis

Neurofibroma.

Dr. Badger's Diagnosis

Chondroma of the lung, or
Calcified hematoma in the region of the posterior end of the sixth left rib, or
Neurofibroma.

Anatomical Diagnosis

Cavernous hemangioma of the left sixth rib.

Pathological Discussion

Dr. Tracy B. Mallory: An interesting phenomenon, which was apparent as soon as the tumor was removed, was that the resected specimen was only half the size of the specimen in the body, as judged by the x-ray films. In other words, it had shrunk 50 per cent during resection, and the reason became obvious as soon as we cut across it. It consisted of a great mass of dilated blood vessels, with small spicules of bone between them. Its gross and microscopic appearance was characteristic of a cavernous hemangioma.

Dr. Hampton: Was there no neurofibromatous tissue in it? It could not be called a vascular neurofibroma?

Dr. Mallory: No.

Dr. Maurice Fremont-Smith: Has anything happened to the neurologic symptoms?

A Physician: No; we have not yet explained the symptoms in his arm except to guess they were due to the proximity of the tumor to the sympathetic chain.

Dr. Kubik: I do not see how that could be the explanation, but the patient may have had another hemangioma, perhaps a very small intraspinal one, which would not necessarily produce any spinal-fluid changes.

CASE 27342

Presentation of Case

A fifty-three-year-old housewife was admitted to the hospital in a semicomatose condition.

Two weeks before admission, the patient was taken ill while in Florida with general malaise and fever of 99 to 102°F. Her condition was diagnosed and treated as "grippe," and the pa-

tient apparently improved, since she was able to ride home by automobile three days later, arriving home one week before admission. During the next two days, she felt well enough to do some housework, but four days before admission she again had general malaise and fever of 102°F. The following day, she developed a slight cough and a stiff neck, and her temperature was 101°F. She was seen by her physician, who found nothing on physical examination except a stiff neck. The day before admission, she felt much better, and her temperature remained normal. On the day of admission, fever recurred, reaching 105.6°F. A sputum examination showed large numbers of Type 8 pneumococci. A white-cell count was 19,000 with 84 per cent polymorphonuclears. The patient was given two 15-gr. doses of sulfathiazole, and in the evening, after being seen by a consultant, was given 30 gr. more and then admitted to the hospital.

The patient had had measles, followed by pyelitis, ten years before admission.

Physical examination showed a well-developed and well-nourished, semicomatose woman who was mumbling slightly, with rare coughing spasms, and who resisted being disturbed. The skin was dry, and the pupils were equal and regular and reacted to light. There was slight but definite rigidity of the neck. The heart did not appear to be enlarged. The sounds were of poor quality, but regular; there were no murmurs. The lungs were clear throughout. The abdomen was soft; no masses were felt. The reflexes were normal throughout. The blood pressure was 84 systolic, 56 diastolic.

The temperature was 105.6°F., the pulse 140, and the respirations 40.

Examination of the blood showed a red-cell count of 5,400,000 with a hemoglobin of 85 per cent, and a white-cell count of 18,000 with 84 per cent polymorphonuclears. Eighteen hours after admission, the white-cell count had risen to 30,000. A catheter specimen of urine showed a specific gravity of 1.028, a + test for albumin and some white blood cells and many finely and coarsely granular casts. A blood culture was negative. The nonprotein nitrogen of the blood was 55 mg., and the blood urea nitrogen 29 mg. per 100 cc. A lumbar puncture showed normal dynamics, and the spinal fluid was normal. A Widal test was negative, and cultures of the stools were negative for typhoid and dysentery organisms. An x-ray film of the chest, taken with a portable machine, was negative.

On admission, the patient was given 5 per cent glucose and physiologic saline solution by clysis, and subsequently, intravenous, rectal and subcutaneous fluids to maintain a daily intake of from 2000 to 3000 cc. Sulfathiazole, 15 gr. every four hours, was continued for the first eighteen hours of the hospital stay but was then omitted. The patient gradually became more responsive, and about twenty-eight hours after admission the temperature had dropped to 101°F. and gradually from there to normal. The pulse dropped only to 110, and the respirations to 25. During the first twenty-eight hours, the urine output by catheter was only 120 cc., but it was believed that the patient voided unmeasured amounts with coughing and with bowel movements.

On the third day, the patient was talkative but very weak. The blood pressure was 86 systolic, 48 diastolic. The cough had become less frequent, but the diarrhea continued. During the next two days, her blood pressure continued to be low, but her cough and diarrhea almost disappeared. On the sixth day, the abdomen was slightly distended and tender in the right hypochondrium. Catheterization yielded 400 cc. of dark turbid urine containing many white blood cells and casts. The next day, the temperature was normal and the pulse 104, but the patient became very lethargic. There was persistent tenderness in the right hypochondrium, and bilateral ankle clonus was found. On the eighth day, she could be roused very slightly; catheterization yielded only 100 cc. of urine in eighteen hours, after an intake of 3000 cc. The patient was given an ampule of 50 per cent glucose solution intravenously. The nonprotein nitrogen of the blood rose to 80 mg. per 100 cc., and the following day, immediately after catheterization, — only 100 cc. of urine had been obtained in the previous fifteen hours, — she had sudden respiratory failure and expired.

Differential Diagnosis

Dr. Earle M. Chapman: Why should an apparently healthy woman of fifty-three years have a minor illness in Florida and then come home to die in anuria about ten days after the return of symptoms?

The illness in Florida was called "grippe," but a sudden fever of 102°F. at least suggests that she might have had malaria. However, there are several points that I should like to rearrange to fit a clinical pattern consistent with an acute infection in the urinary tract. First is the fact that she had pyelitis ten years before admission — a disease known to recur when people suffer fatigue and dehydration. Certainly, the environment in Florida and during the drive back home was conducive to both factors. Likewise, the ir-

regular fever, the high white-cell count and the vague symptoms are compatible with pyelitis or even pyelonephritis. Unfortunately, there is no report of the urine at this period of her illness.

Evidently, the single finding of a Type 8 pneumococcus in the sputum led her physician to suspect a pulmonary infection. Although Type 8 ranks about the fifth or sixth most frequent type found in persons with pneumonia, it could well be a harmless inhabitant of the nasopharynx in a person returning from Florida to a New England winter.

With a suspected pulmonary infection and with stiffness of the neck and a semicomatose condition, one thinks of a brain abscess secondary to a diseased lung. Again, we cannot substantiate any such diagnosis by physical examination, chest x-ray film or lumbar puncture; and, of course, we know that sulfathiazole is poorly concentrated in the cerebrospinal fluid and is therefore contraindicated in cases of meningitis or brain abscess.

After receiving 4 gm. (60 gr.) of sulfathiazole, the patient was much worse and was admitted to the hospital in rather poor condition. She was comatose, coughing and behaving in a peculiar manner. There is no description of the scleras or conjunctivas. It is significant that she already had nitrogen retention and a urine containing albumin, white blood cells and granular casts. Adequate fluids were given, but still she did not excrete a proper quantity of urine. Something was evidently wrong — the sulfathiazole was stopped after a total of 8 gm. had been given in a period of about forty-eight hours. Then she seemed to improve, and the temperature returned to normal. However, the pulse continued to be rapid, and the blood pressure became even lower in its maintained or diastolic level. Then there was a cessation of the diarrhea — which explains the need of the previous laboratory studies that were negative.

The signs of kidney failure increased — the urine became turbid and dark and contained many white blood cells. Congestive failure, or some toxic effect, caused distention and tenderness of the liver. Lethargy, drowsiness and coma supervened, and the nonprotein nitrogen reached 80 mg. per 100 cc.; yet the blood pressure remained low. Concentrated glucose did not bring about a diuresis. She died of sudden respiratory failure. I doubt that she had a silent occlusion of a coronary artery as a cause of this.

It is my belief that the patient died in anuria because her kidneys, previously diseased and possibly showing pyelonephritis, did not excrete sulfathiazole properly. It would be helpful to know the blood level of the drug for the several days after it was stopped. Sulfathiazole is definitely more toxic to the kidneys than the other sulfonamide drugs are. It seems that the kidney tissue was particularly sensitive in this woman, since we have no description of the commonest signs of toxicity — an erythematous skin lesion or reddened conjunctivas. The fever, leukocytosis, diarrhea and mental changes could be part of the reaction. I am not surprised by the absence of anemia. The stiffness of the neck does concern me, and unless we call it meningismus, I cannot explain it.

From reported cases of anuria caused by sulfapyridine and sulfathiazole, we know that the chief difficulty is with a plugging of the collecting tubules, or even the ureters at any level, by the drug crystals and secondary detritus. Consequently, microscopic sections have shown great distention and plugging of the tubules, while the glomeruli remained comparatively normal. If such a plugging had occurred in this case, we could expect that cystoscopy and ureteral lavage would have been therapeutically effective.

Dr. Fletcher Colby: That seems to be a good possibility. On the other hand, is it not quite possible that she just had chronic nephritis of such severe degree that a superimposed acute infection was enough to produce anuria without implicating sulfathiazole?

Dr. Alfred Kranes: The specific gravity of 1.028 is somewhat against chronic nephritis.

Clinical Diagnosis

Bronchopneumonia?

Dr. Chapman's Diagnoses

Chronic pyelonephritis.
Sulfathiazole toxicity, with plugging of ureters and tubules of the kidneys, leading to anuria.

Anatomical Diagnoses

Acute pyelonephritis, bilateral.
Pericarditis, acute.
Myocarditis, acute.
Pulmonary edema and congestion.
Ascites, slight.

Pathological Discussion

Dr. Benjamin Castleman: This patient was studied at another hospital, but the autopsy was performed by one of our laboratory staff. Clinically, because of the finding of the Type 8 pneumococcus in the sputum, a great deal of attention was paid to her chest, and it was believed that she

probably had bronchopneumonia. The autopsy, however, showed no evidence of pneumonia, just a little edema and congestion of the lungs. Her kidneys were large, weighing 385 gm. The capsules stripped fairly easily, and the surfaces showed a reddish mottling and few grayish-white spots that suggested small abscesses. The sections microscopically showed a diffuse acute pyelonephritis. The tubules were filled with red and white cells, and there were a number of small abscesses throughout the kidney. We specifically looked for sulfathiazole crystals and could not find any, either grossly or microscopically. The severe degree of pyelonephritis, however, was enough to have produced the renal insufficiency. We have had this year, I believe, four cases of acute pyelonephritis, very similar to this, in which the signs and symptoms did not particularly point to the kidney. The only clue here was the presence of leukocytes in the catheterized specimen. Unfortunately, the urine was not cultured. There was about 50 cc. of turbid fluid and a few shreds of fibrin in the pericardial cavity, and sections of the myocardium showed a few collections of polymorphonuclears. Bacteriologically, we were able to grow nonhemolytic streptococci from the kidney and spleen.

DR. JOHN H. TALBOTT: Did the patient have a cerebral abscess?

DR. CASTLEMAN: The brain was not examined.

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MATERNAL WELFARE AND THE NATIONAL EMERGENCY

World War II is inevitably forcing the country to face a national emergency in maternity care. Already the armed forces have taken large numbers of physicians into service, and future estimates call for nearly half of today's active practitioners. This need must be met. But equally so, maternal care must not be jeopardized. The totalitarian countries have been quick to realize this, for their armies of the future can exist only in the babies of today. To them, the mother and her child are irreplaceable assets. One must hope that in lands granted the opportunity of building for a peaceful, productive future, maternal welfare will be no less zealously guarded.

The problem in this country is not a new one. Ever since the introduction of routine prenatal examinations in the early years of this century, more and more attention has been paid to organized maternal care. The result has been a gratifying but by no means satisfactory reduction in maternal deaths and morbidity. Even in 1939, there were over 12,000 live births without medical attention. Less fortunate still were those women cared for by practitioners of meddlesome obstetrics. It is clear that further decrease in mortality and morbidity rates will come only through an improvement in the quality of obstetric care and in its extension to the entire population. Ideally this should be provided by a sufficiently large number of physicians well trained in obstetrics. An all-out program of this sort would include a more thorough undergraduate obstetric training, adequate postgraduate educational opportunities, adjunct obstetric nursing services, improved hospital facilities, lay education, consultation services, and an organization of medical facilities to bring all economic and geographic units within the scope of the program. Thus, every pregnant woman would be cared for by a physician basically well-trained in obstetrics, with all possible specialized assistance at hand whenever needed. This goal has never been reached; when it may be attained in the future one cannot foretell. With the removal of ever-increasing numbers of physicians from civilian life the date will be further postponed. But the problem is one of the immediate present. In solving it, America might do well to turn to the midwife. Her help now and in the reconstruction to follow would be invaluable; it is probable that in certain capacities she would become part of the permanent obstetric service of the nation.

Although the midwife never has been much favored in the United States, she still delivers more than half the babies of Europe. She has played her part in the notably low mortalities of the Netherlands and Scandinavia. Midwifery services in the United States as well have demonstrated that they can offer safe pregnancy care. The Frontier Nursing Service of Kentucky covers

seven hundred square miles of territory in mountainous Leslie County and has delivered over 3000 babies, with a gross uncorrected maternal mortality of 0.68 per 1000 live births — the maternal mortality rate for the country as a whole is approximately 4 per 1000. The Philadelphia Midwives Service shows a mortality rate of half that of the city as a whole. Louisiana has reduced its maternal mortality significantly through obstetric care offered by its colored midwives. The Outpatient Department of the Boston Lying-in Hospital, manned by externs, has a rate of 0.15 per 1000 home deliveries: this and similar departments elsewhere are basically midwifery services. It must be noted, however, that these results have been obtained only with carefully and rigidly controlled systems under competent medical supervision, with proper hospital facilities in the background. Furthermore, it is clear that the best figures come from the services staffed by the modern type of midwife.

The new order of midwife is a graduate nurse who has had additional formal training in obstetrics. In training, therefore, she stands between the obstetric nurse and the obstetrician. She may hold the certificate of the Central Midwives Board of Great Britain or be a graduate of the Lobenstein School, of New York City, or of the newly founded school of the Frontier Nursing Service; she will undoubtedly find other schools offering similar courses. She is licensed as carefully as the physician. Over her and working with her are competent consulting obstetricians, and always there is access to a hospital. Cases are registered early with her service and are selected according to anticipated difficulties. Patients with complications are hospitalized before they become serious problems. Emergencies are met by well-ordered teamwork and adequate facilities. The important phase of lay education is handled through individual conferences, mothers' and fathers' clubs, and other groups. Thus, the midwife is not expected to handle cases beyond her ability, and the consulting obstetrician can offer his highly trained services over a much wider area. From the point of view of efficiency, no other type of maternity service can bring competent care in its fullest sense to so large a number, either in crowded city tenements or in sparsely settled, inaccessible country regions; hence, it seems reasonable to recommend that the further development of this new type of midwifery receive due consideration.

A MUNICIPAL INSTITUTE FOR MEDICAL RESEARCH

The Health Commissioner of New York City has announced the organization of the Public Health Research Institute, to be operated by the city with a budget of $100,000 for 1941–1942, and to be administered by the Health Department, under a research council and a board of directors. The members of the Research Council and the Board of Directors will all serve without salary. The council will be responsible for the scientific personnel, and the board will assure sound business management of the institute. This is apparently the first time a city government has undertaken an enterprise in medical research "for the protection and the improvement of the health, safety and welfare of the people."

The initial validity and success of the institute is vouchsafed by the membership already chosen for its research council and board of directors. As time passes, however, these members must necessarily be replaced, and its future depends to a very great extent on the type of persons who are available and chosen to succeed. Moreover, the institute must be guarded against becoming just another city bureau that justifies its existence by an adequate routine and continues its life through the virtues of the annual budget. It also needs to be guarded against scientific overenthusiasm, which might carry its efforts far afield from the items of importance to modern city dwellers.

There is plenty yet to be done for the city dwellers of this country. Their lives are still shorter than those of the rural dwellers — a fact discreetly avoided by those who have been promoting "socialized medicine" under the agricultural aegis. An establishment of the precise reasons why this is so might be one of the earlier proj-

ects to which the institute can address itself. The city dweller is well protected against polluted water, infected food and incompetent systems for sewage disposal, but he is allowed less sunlight, is forced to inhale more foreign materials, is exposed to a greater variety of respiratory viruses and probably subjects himself to a greater and more frequent climatic variation than does the man on the farm. The health authorities of all our large cities are also now concerned with housing, with transportation facilities, with transient populations and with an ever-widening field of application for the knowledge and understanding of environment.

Starting out with a relatively modest budget, the institute will have a splendid opportunity to prove itself. Provisions have been made for it to receive and accept grants, gifts, bequests and contributions from private citizens and foundations. Its future scope is thus not limited or altogether dependent on the expediences or political necessities that have been known to cramp many other governmental enterprises. If the largest city in the world finds that a public-health research institute is of value, other cities will surely follow suit, for the largest city has no monopoly on municipal problems concerned with the health and welfare of the citizens. It is hoped that this new type of research motive will prove successful.

MEDICAL EPONYM

Joffroy's Sign

"Nature et traitement du goitre exophtalmique [The Nature and Treatment of Exophthalmic Goiter]" was the subject of a lecture delivered by Alexis Joffroy (1844–1909) in December, 1891, in the neurological clinic of the Hospice de la Salpétriere. This lecture appears in *Progres médical* (18: 477–480, 1893). A portion of the translation follows:

> *Paralysis of the muscles of the upper part of the face.* In our patient, these muscles were affected in a peculiar way that I have seen previously in three other cases, although this peculiarity has never been pointed out before. It is as follows: if a person who is looking downward is asked to look up at the ceiling quickly, holding his head still, it will be found that as the eyeballs roll upward, the eyebrows are raised and the forehead wrinkles. We have here a synergic movement occurring in the normal state that does not occur in this patient. Her eyebrows and forehead remain absolutely immobile, even when she tries very hard to look upward.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Fatal Sepsis Following Abortion

A twenty-seven-year-old para V, approximately three months pregnant, was admitted to the hospital with a high temperature and with a catheter protruding from the vagina.

The past history contained no pertinent details. The patient had apparently had no prenatal care. Physical examination was essentially negative except for the elevated temperature, a corresponding rise in pulse rate, and tenderness and spasm over the entire lower abdomen. The patient was having cramplike pains, and a few hours later delivered herself spontaneously of a macerated fetus and the placenta. Following delivery, she had a chill, which lasted ten minutes. The temperature ran a high septic course, and the patient developed jaundice. The treatment consisted in several transfusions, the uterus being left entirely alone. Death occurred on the eleventh day after admission.

Autopsy revealed extensive jaundice; the uterus was necrotic from the cervix to the fundus, the spleen was enlarged to twice its normal size, the left ovary and tube were necrotic, and the right ovary and tube were infected.

Comment. There is nothing in this history to reveal whether this was a criminal abortion or one brought on by the introduction of a catheter by the patient herself. When she arrived at the hospital, she was septic, and she died of profound sepsis ten days later. The jaundice was characteristic of that sometimes associated with infection, and the infection of the uterus, tubes and ovaries was typical of the sepsis that often follows abortion. This death occurred before the introduction of the sulfonamide drugs, and it is possible that intensive chemotherapy might have averted this death.

As in many of these cases, this patient was probably treated by an abortionist. The end result is one that may follow any criminal abortion — a tragedy that, if generally recognized, might prevent some women from looking on this procedure with perfect nonchalance.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

DEATHS

ALBEE — GEORGE M. ALBEE, M.D., of Worcester, died August 10. He was in his seventy-first year.

He received his degree from the New York University Medical College in 1893, and had practiced in Worcester for many years, having been particularly interested in cardiology.

Dr. Albee was a fellow of the Massachusetts Medical Society and the American Medical Association. He was also a fellow of the American College of Physicians, and a member of the New England Heart Association and the American Heart Association.

LORD — WILLIAM O. LORD, M.D., of New Bedford, died August 8. He was in his sixty-first year.

Born in New Bedford, he received his degree from the Columbia University College of Physicians and Surgeons in 1903.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a son, a half-brother and two half-sisters survive him.

REPORT OF MEETING

NEW ENGLAND PATHOLOGICAL SOCIETY

A regular meeting of the New England Pathological Society was held on April 17, Dr. Tracy B. Mallory presiding. Dr. Arthur Purdy Stout, associate professor of surgery at Columbia University and attending surgical pathologist to the Presbyterian Hospital in New York City, spoke on "Tumors of Peripheral Nerves." Dr. Stout divided the tumors of the peripheral nerves into six groups (Table 1), and the discussion dealt chiefly with the question of the cellular origin and the composition of the primary neoplasms. The work of Masson and Nageotte and the tissue-culture observations of Dr. Margaret Murray suggest very strongly that the dominant tissue of the nerve sheath is the neurectodermal schwannian syncytium. This grows in a characteristic fashion in vivo and in vitro, and can form reticulin fibers. The fact that the schwannian cells of the nerve sheath can form connective-tissue fibers need cause no surprise, since it is well known that the cells of the neural crest form connective tissue (mesectoderm) in the human embryo. In urodeles, this mesectoderm forms adult connective tissue and cartilage.

These observations form a solid foundation for the hypothesis that most of the primary nerve-sheath tumors are of neurectodermal origin and composition. They explain the connective-tissue fibers of the neurilemmoma, neurofibroma and malignant neurilemmoma (schwannoma) and the bone and cartilage of the teratoid tumors of peripheral nerves. Further confirmation comes from the fact that Dr. Murray has grown almost pure cultures of schwannian cells from neurilemmomas and neurofibromas and from one case of malignant neurilemmoma (schwannoma). The formation of adult ganglion cells by ganglioneuromas and of true rosettes by neuroepitheliomas establishes their neurectodermal origin. The neurectodermal origin of the pigmented skin tumors is suggested by the observations of Masson and Laidlaw, but the speaker did not accept it as proved.

According to Dr. Stout, primary neoplasms of purely mesodermal origin include hemangiomas and lipomas. Whether ganglions, neuroxanthomas, fibromas and so-called "neurogenic sarcomas" are true nerve-sheath tumors of mesodermal origin is open to debate. He denied that there is adequate proof that ganglions (similar to the familiar ganglions of the wrist), fibromas and xanthomas ever grow in or from peripheral nerves. Proof is also lacking that any of the malignant spindle-cell tumors that form connective-tissue fibers and grow in or from the peripheral nerves are of mesodermal origin. Therefore, until such proof is forthcoming, the use of the term "neurogenic sarcoma" is not justified.

The discussion was opened by Dr. Orville T. Bailey, who confessed his bewilderment concerning the histogenesis of the nerve-sheath tumor. He believes that new methods of study must be developed before any decision can be made; he stated that the work of Dr. Stout and Dr. Murray had opened up new possibilities that may do a good deal toward clarifying the situation. He pointed out that there is one problem, without the solution of which one cannot consider the problem of histogenesis completely solved — namely, the occasional resemblance of nerve-sheath tumors to meningiomas. Of course, both types of tumor vary a good deal in histologic structure, and if one acquires a large series of both tumors and arranges them in a graded series, the two extremes are very different; there is an overlapping stage, however, indicating that somehow or other the problem

TABLE 1. *Tumors of Peripheral Nerves.*

NON-NEOPLASTIC NEURECTODERMAL TUMORS	MESODERMAL NEOPLASMS
Traumatic or amputation neuroma	Hemangioma
Spontaneous neuroma	Lipoma
Appendiceal neuroma	(Ganglion?)
	(Neuroxanthoma?)
NEURECTODERMAL NEOPLASMS	(Fibroma?)
Neurilemmoma	("Neurogenic [neurogenous] sarcoma"?)
Neurofibroma	TUMORS AND LESIONS WHOSE GROWTH IS INDUCED BY NEUROGENOUS TUMORS
Ganglioneuroma	Fibroma molluscum
Neuroepithelioma	Multiple adenoma sebaceum
Malignant neurilemmoma	Elephantiac hypertrophy of bone, intestine, appendix and so forth
Teratoid tumor	Hypertrichosis
(Pigmented mole and melanoma)	Skin pigmentation
(Mesectodermoma?)	Multiple lipomas
NEOPLASMS COMPOSED OF MULTIPLE TISSUES OF WHICH NEURAXONS FORM ONE IMPORTANT ELEMENT	SECONDARY NEOPLASMS
Glomus tumor	Direct extension within nerve sheath
Cutaneous leiomyoma	Intraneural metastasis
	Tumors of ganglia lying within nerves

of the histogenesis of the two is related. Also, the appearance of multiple meningiomas in patients with multiple neurofibromatosis is another indication of their possible relation. If one has only a small fragment of tissue to examine, it is possible to find areas that are almost entirely composed of schwannian cells in the ganglioneuromas occurring outside the central nervous system. The occurrence of these areas was very striking in a case reported by Cushing and Wolbach some time ago, in which the diagnosis for a long time was obscured by this difficulty. Dr. Bailey believes that the description of the neuroxanthoma suggests strongly that one of the forms of sclerosing hemangiomas is involved. Some hemangiomas are associated or occur in the immediate vicinity of pigmented nevi, and the formation of giant cells and lipoid phagocytes is a part of the sclerosing process that goes on in many hemangiomas. Furthermore, this explains very well the histologic appearance of infiltration. The hemangioma has not infiltrated surrounding tissue, but adjacent connective tissue has been stimulated to form its stroma. The glomus tumors, according to Dr. Bailey, include a large number of nerve fibers that are the integral part of the tumors themselves. In addition, they have glomus cells that appear to be derived from the smooth-muscle elements of the normal glomus. The combination of these fibers and cells not only results in a complex tumor from the morphologic point of view but actually takes on the function of a gigantic glomus.

Dr. Stout commented on Dr. Bailey's discussion and said that Dr. Murray had also been attempting a study of meningiomas from a tissue-culture point of view. There are different kinds of meningiomas, and they show quite different characteristics. Cultures of what seem to be meningeal schwannian-cell tumors resemble cultures of schwannian peripheral-nerve tumors; they are otherwise quite different. Dr. Murray has also had an opportunity to grow three glomus tumors, and they have been extremely interesting in tissue culture because they have grown out endothelium that could be recognized quite easily in vitro. They have also grown out schwannian cells, which is to be expected because there are so many nerves in the glomus tumors. Finally, they have grown out the so-called "epithelioid cells." These have done a very interesting thing in culture. When endothelium has grown out in long thick syncytial masses, the epithelioid cells of the glomus tumor attach themselves in clusters to the endothelium. Therefore, the relation of the epithelioid cells to the smooth muscle cells that Dr. Bailey spoke about is very possible. Drs. Stout and Murray were fortunate enough to get hold of an infiltrating glomus tumor and grow it in vitro, and it behaved in exactly the same way as the benign glomus tumors. Dr. Stout had never before come in contact with an infiltrating glomus tumor, but apparently it was genuine.

Dr. Robert Gross continued the discussion by stating that in the classification of the neuroectodermal tumors Dr. Stout mentioned neurilemmoma and malignant neurilemmoma. Dr. Gross believes that the malignant neurilemmomas might be further divided, because in reviewing the specimens at the Peter Bent Brigham and Children's hospitals, it was evident that some have more malignant qualities than others, so far as the clinical characteristics are concerned. He said that the benign tumors are called "neurilemmomas," the middle group "malignant neurilemmomas," and the more malignant ones "neurogenic sarcomas," although he acknowledged that these terms are incorrect, in comparison with their use by other pathologists. In the middle group, the findings are not the same as those in the rank and file of sarcomas. This type of neoplasm tends to recur locally but does not metastasize. It can be recognized histologically in that the cells have more cytoplasm than those of the neurilemmoma; the fibers are shorter; the nuclei are vesicular, and they may have occasional mitotic figures. Dr. Gross stated that he had seen four of these tumors. One, of the fifth nerve, was in a thirty-two-year-old woman who had recurrences in spite of five or six attempts at removal and heavy x-ray therapy. Another, in a thirty-year-old woman with a tumor of the left subscapular nerve, which was locally excised, recurred, and two years later a further excision was done. This went on for fourteen years before massive local recurrence extended into the thorax. The third patient was a child with a lesion of the little finger. The tumor recurred after six local operations, and finally ended with removal of the ulnar half of the hand. The last case encountered was that of a twenty-three-year-old man with an egg-sized tumor of the posterior cord of the brachial plexus. Since, histologically, this tumor had the same characteristics that were used for placing neoplasms in this group of malignant neurilemmoma, local excision by peeling it out from the cord was not done, but the entire posterior cord was removed. There has been no evidence of recurrence. Dr. Gross considers it essential to recognize this group because of the practical considerations involved. In other words, these tumors do not need to be treated by amputation, as neurogenic sarcomas that occur in extremities do, and conversely cannot be treated properly by enucleation, as one does in a benign tumor (neurilemmoma), by peeling it out from the sheath. Therapy must lie between the more radical and conservative forms; in other words, the entire mass, with nerve trunk, must be excised.

Dr. Stout admitted that the classification of malignant neurogenous tumors used is not sufficiently complete. He thinks that it is probably necessary to subdivide the malignant neurilemmomas, or schwannomas. He added that most of these, as Dr. Gross had said, are malignant by infiltrative growth, and very few of them metastasize. Dr. Stout is as yet unable to subdivide them into grades of malignancy.

Dr. Donal Murnaghan, of Ireland, stated that a few days previously Dr. Murray had very kindly shown him some of her preparations; some were living, others fixed and stained. His own experience, from studies made in the laboratory of Dr. Warren Lewis in the Carnegie Institute of Washington at Baltimore, was in complete agreement with her identification of schwannian cells. He recognized the same type of cell in chick-embryo and mouse-embryo material and noted the similarity between them; the satellite cells of spinal-ganglion cells were tentatively identified as oligodendroglial, the latter in cultures of the central nervous system. In cultures of embryonic peripheral nerves, rapid outgrowth of mesenchymal cells soon obscured the migrating schwannian cells, and he did not follow them. His experience did not extend to adult tissues, and therefore he did not express an opinion on Dr. Murray's work in this field, except to acknowledge her ability to distinguish schwannian cells from fibroblasts. Embryologists believe that oligodendroglia and schwannian cells arise from equivalent sources in the neural ectoderm, a term that should be used with reservations because it is now believed that the time-honored, three-germ layer is more a convenient than an accurate representation of the true state of affairs in the young developing embryo. Histologists are familiar with the similarity between spinal-ganglion satellite cells and the sheath cells

of Schwann, and now cytologists find certain morphologic and cultural characteristics common to all three. He stated that oligodendroglia, the satellite cells of spinal-ganglion cells and schwannian cells, are morphologically equivalent, showing similar characteristics, embryologic, structural and cytologic. He then showed in a motion-picture film how oligodendroglia grow out in a culture of the midbrain of a seven-day chick embryo, in a fluid medium, and to indicate the method of locomotion so beautifully demonstrated by some of these cells.

Dr. Tracy B. Mallory continued the discussion. He said that a pathologist is often called on by the surgeon to give an opinion regarding therapy in local recurrence, since it is known that some tumors metastasize. Often, a patient comes in who has had one or two or even three recurrences of definite malignant types. The question comes up whether amputation or another local excision should be done. He asked Dr. Stout to say a little more about treatment, about radiation of the malignant type and also how the acoustic neuromas fitted into his classification.

Dr. Stout answered that if the tumor is a malignant form occurring in von Recklinghausen's disease, practically none have been cured either by excision or amputation. Possibly the reason for that is that von Recklinghausen's disease is a generalized disease in which many nerves are involved, and if one succeeds in removing one of them that seems to be malignant at the moment, the tendency to form malignant tumors in that patient is great enough for other tumors to form further progressive growths. When they occur without von Recklinghausen's disease, the indication is to try to get rid of the local growth. If its extent is so wide as to necessitate amputation, one should not hesitate to advise amputation. From personal experience and reading, Dr. Stout believes that none of these tumors are radiosensitive, and that none are destroyed by irradiation. The very rare neuroepitheliomas are extremely malignant metastasizing tumors, and the most radical form of treatment is indicated.

Dr. Stout remarked that he had seen fewer acoustic tumors than tumors farther away from the central nervous system. Those that he had seen appeared to be neurilemmomas. They show some variance and have a much greater tendency to have cells containing lipoid than the peripheral tumors, in which lipoid is only occasionally found. He said that Dr. Murray has grown three angle tumors in vitro, and that they, too, grew out cells that were interpreted as schwannian cells.

BOOK REVIEWS

Radiologic Physics. By Charles Weyl, M.S.; S. Reid Warren, Jr., Sc.D.; and Dallett B. O'Neill. With a foreword by Eugene P. Pendergrass, M.D. 8°, cloth, 460 pp., with 166 illustrations and 33 tables. Springfield, Illinois: Charles C Thomas, 1941. $5.50.

This book deals with those branches of physics that are met with in the study and application of radiant energy. It also covers, but to a lesser extent, the other types of electrical apparatus found in the modern hospital. As noted in the introduction, the book attempts to bridge the gap between no knowledge of physics and advanced specialization. To the reviewer, such a bridge is too long for a single span. The major portion of the book contains material that is too far advanced for the average practitioner or student of radiology, who has no special bent for the physical problems related to his specialty, and it is apparently designed for the person who is doing research work in this subject. The first two chapters of the book, on the other hand, deal with material encountered in the most elementary courses of science in high schools, and there is an appendix of forty pages containing the elements of algebra, trigonometry and calculus.

There is a good discussion of the methods of measurement of quantity and quality of radiant energy, but too brief a discussion of the use of metal filters. Only a part of one chapter has been devoted to discussion of the circuits commonly used in x-ray generators, and there are only a few remarks concerning the advantages and disadvantages of the various types.

On the whole, the book seems not to provide a satisfactory source of information for the average student of radiology who wishes to know something of the machines with which he works.

Blood Transfusion Association Report: Concerning the project for supplying blood plasma to England, which has been carried on jointly with the American Red Cross from August, 1940, to January, 1941. 8°, paper, 121 pp. New York: Blood Transfusion Association, 1941.

A blood-transfusion association has been operated in New York City since 1929, with the principal object of improving the supply of blood for transfusion purposes in that city. In June, 1940, in conjunction with the American Red Cross, the association greatly expanded its work to supply blood plasma for England. In a period of six months, its members collected over 5000 liters from about 15,000 persons, and much of this plasma was sent to England. After the first of January, 1941, England was able to supply its own needs, and the organization closed down this part of its work. Wisely, however, research concerning the advantages and disadvantages of blood plasma or blood serum, both in the liquid and the dry states, has been continued, and this work is to be carried on until more definite conclusions can be reached. The report of this six-month endeavor contains much valuable information about the organization of the work, and is a medical report of great value. It is a welcome addition to the literature.

Applied Physiology. Oxford Medical Publications. By Samson Wright, M.D., F.R.C.P. Seventh edition. 8°, cloth, 787 pp., with 367 illustrations and 5 color plates. New York: Oxford University Press, 1940. $7.00.

Since the first edition of this commendable textbook, published in 1926, the author has been consistent and successful in his aim to present the facts of physiology for use by men working in the wards and various other departments of hospitals. This new edition has been drastically revised by the incorporation of recent advances in this science. As a member of the editorial staff of Section A-III of the *British Chemical and Physiological Abstracts*, the author was particularly well placed for this task.

The book has been enlarged, and contains five colored plates and many illustrations. Students will find many cross references of recent investigations, inserted throughout the text. It may be noted that the author has not lost sight of the academic aspect of physiology in his emphasis on its applied aspects.